THE PLANT DISEASE REPORTER

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THE PLANT DISEASE SURVEY

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BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING
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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

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IN THIS ISSUE

George W. Fischer and L. J. Tyler give evidence suggesting earlier occurrence of dwarf bunt than 1931, the year in which it was first recorded from widely separated localities, page 445.

Septoria black-stem and kernel blight of oats does not appear to be seed-transmitted, according to experimental results reported by M. D. Simons and H. C. Murphy, page 448, nor was infected seed toxic to chickens and mice in limited feeding tests.

H. H. McKinney and others find that nutrition is a major factor in chlorophyll degeneration in oats and grasses, page 450.

Pen Ching Cheo and W. J. Zaumeyer, page 459, describe a new severe strain of tobacco ringspot virus isolated from mottled bean pods collected in Virginia and Delaware.

R. H. Fulton, page 466, reports that the strawberry type 2 virus in the Robinson variety was not destroyed by heat treatments that could be endured by the plants tested.

R. O. Magie et al. report the presence of the western aster-yellows virus in Oregon-grown gladiolus corms planted in Florida, page 468.

Hurley Fellows and Richard V. Connin, page 471, describe a glass and plastic insect-proof cage for experimental work, particularly with virus transmission.

The occurrence of Curvularia on gladiolus in Oregon is reported by C. G. Anderson et al., page 474.

Soil fumigation with chlorobromopropene reduced the amount of Sclerotium rolfsii in the soil, but on the other hand thorough fumigation also permitted other inadvertently introduced pathogens to increase, apparently by destroying inhibitors; the experiments, reported by Frank A. Haasis, page 475, were for the control of Sclerotium on Dutch iris in North Carolina.

W. C. Barnes and Wm. M. Epps, page 479, report evidence indicating two types of anthracnose resistance in cucumbers.

A transit and storage rot of California peppers caused by Cladosporium herbarum is described by G. B. Ramsey and Barbara C. Heiberg, page 481.

The Long Island golden nematode of potatoes apparently will not develop on tobacco, although the larvae enter the roots, according to experimental results reported in two articles, by Julius Feldmesser and George Fassuliotis, page 483, and by W. F. Mai, page 485.

R. D. Watson and Ted Gessel describe lightning injury to sugar beets as observed in Idaho, page 487.

Oak shade trees with roots extensively rotted and weakened by Polyporus dryadeus are a serious windthrow hazard, according to W. A. Campbell and J. H. Miller, page 490.

Unusual records and other brief notes, page 491, include reports of soybean target spot in Virginia, by Helen S. Sherwin; severe and widespread occurrence of a virus disease on Ladino clover in Virginia, by S. B. Fenne; and miscellaneous notes from Texas, including a report of Pratylenchus zeae, by A. L. Harrison. Also, J. A. Stevenson describes the literature indexes of the Division of Mycology and Disease Survey.

Announcement: the Plant Protection Bulletin of the Food and Agriculture Organization of the United Nations, page 492.

J. A. Stevenson reports the appearance of the revised edition of the rules of botanical nomenclature, page 492.

Attention please! Especially for contributors, page 494.

October weather, page 495.

PLEASE NOTE: Copy for illustrations for the Reporter should be sent unmounted (See PDR 36(5): 214, May 15).

Vol. 36, No. 12--PLANT DISEASE REPORTER--Dec. 15, 1952

IS THERE AN EARLIER RECORD OF DWARF BUNT THAN 1931?

George W. Fischer and L. J. Tyler[1]

The first published report of dwarf bunt (Tilletia brevifaciens G. W. Fisch.) of wheat, rye (Woodward, Holton and Vogel, 1952), and grasses (Fischer, 1952; Hardison and Corden, 1952) was by Young (1935) who had observed and studied the disease in Montana since 1931. Probably the next was later in the same year by Tingey and Woodward (1935) in Utah. The latter authors did not differentiate dwarf bunt from ordinary bunt, but their cover page picture of "covered smut" in Utah Kanred wheat can leave little doubt but that they were dealing with dwarf bunt. In 1936, Holton and Heald first reported this smut from Washington, stating that it had been under observation since 1931. Rodenhiser and Holton (1945) stated further that the disease "was observed in devastating proportions over a fairly wide area in southeastern Idaho and northern Utah" in 1931. Bodine and Durrell (1941) have been credited with the first report of dwarf bunt from Colorado, but they merely reported that "Tilletia tritici was found for the first time in Montezuma, Moffat and Rautt Counties." Other than that they further stated "that the usual seed treatment .... did not give control," there was no identification with the dwarf bunt disease. However, that this report did refer to dwarf bunt was later verified by Rodenhiser and Holton (1945) from specimens submitted to them. The first published record of the disease in Wyoming was by Blodgett in 1944. In 1949, Foster and MacSwan reported its incidence in British Columbia. Dwarf bunt in wheat was not formally reported for Oregon until recently (Hardison and Corden, 1952), although it was officially identified from that State in 1948 by C. S. Holton.

Until 1940, when it was collected in New York State (Tyler, 1945), dwarf bunt was considered to be restricted geographically to the Pacific Northwest and Rocky Mountain States. Recently, Conners and Skolko (1952) have added the neighboring province of Ontario, where dwarf bunt is now known to occur in one district.

Just when dwarf bunt was first detected outside of North America is uncertain. Pichler (1951) claimed that the first detection of the disease in Europe was in Bavaria in 1942. However, Wagner (1950) believed that Fittschen (1939) had dwarf bunt among the bunt races he was using in the testing of wheat varieties for bunt resistance. Fittschen himself (op. cit.) recognized the similarities of his "Linie K" to Young's (1935) "new race of Tilletia tritici" and to the "short smut" of Holton and Heald (1936). Fittschen's "Linie K" originated in Zurich in 1926, and if it should be established that this line definitely was dwarf bunt, we would then have an earlier authentic record for Europe than we have at present for North America. Considerable doubt is cast on this possibility, however, since Fittschen (op. cit.) observed that his "Linie K" differed from the American dwarf bunt (i. e. in gross appearance and behavior) in only one respect: He was able repeatedly to obtain infection by seed inoculation, whereas in all American studies of dwarf bunt even very low percentages of infection have only rarely been obtained by this method.

Thus it would appear that so far the earliest authentic record of dwarf bunt was from Montana in 1931 (Young, 1935). However, Tingey (1950) states that "in the '20's dwarf bunt was already well established in Cache and Box Elder Counties" in Utah. He based his statement on testimony of wheat farmers in the dry land areas of the State. Tingey (op. cit.) also cites circumstantial evidence that this disease may have been present in Utah "even before 1903". Whether these claims of the earlier (i. e. earlier than 1931) occurrence of dwarf bunt can be considered as valid unless substantiated by herbarium or other authentic specimens seems doubtful to us.

In this connection, recent restudy of herbarium specimens of collections of wheat bunt in New York State has revealed that the first official record of dwarf bunt of wheat in that State was in 1931. This is nine years earlier than first believed (Tyler, 1945), and the same year it was first recognized in Montana (Young, 1935) and Washington (Holton and Heald, 1936), and observed "in devastating proportions" in southwestern Idaho and adjacent Utah (Rodenhiser and Holton, 1945). This 1931 collection in New York State is No. 19367 in the Plant Pathology herbarium of Cornell University, collected by M. F. Barrus on "Forward Wheat", July 11, 1931, near Rushville, New York. It had been determined by H. H. Whetzel as "Tilletia tritici" (T. caries). Dr. Barrus still remembers making the collection and tells us that his attention was drawn to this particular incidence of wheat bunt by the unusual dwarfing effect on the host plants. No further special interest was then directed to the collection, however.

A similar experience was related by Conners and Savile (1949) when they confirmed the discovery of dwarf bunt in Canada, where it was reported by Foster and MacSwan (1949) in British Columbia in the Armstrong-Enderby district and on one farm on Vancouver Island. The former

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authors noted that "there exists in the herbarium a specimen of T. caries, collected at Armstrong in 1931, that has subsequently been identified as dwarf bunt." This record established the fact that dwarf bunt existed in British Columbia at least 17 years before it was officially reported.

These two instances of the identification of wheat bunt specimens as dwarf bunt 21 and 17 years, respectively, after they had been collected, reopens a subject of considerable academic interest and perhaps of some practical importance as well. In how many other herbaria and laboratories are earlier collections of dwarf bunt lying unrecognized? The academic interest in this question is a matter of determining as closely as possible just when and where in the world dwarf bunt was first found and the dates of its first incidence in other states and countries after that. The practical implications pertain to determining the relative spread and progressive severity of the disease during the ensuing years after it was first found in the various parts of the world. These data, if correlated with information on local climatic and edaphic factors, could give clues as to the comparative degree in which dwarf bunt has been favored or inhibited in various ecologic and geographic regions by such factors as climate, physical and chemical condition of the soils, various rotation and other cultural practices, and varietal reaction.

It would appear from the foregoing that there are official records of dwarf bunt's being collected or observed in British Columbia, Idaho, New York, Utah, and Washington, all in the same year -- 1931. It does not seem likely that this disease would make its debut simultaneously in all these States and Provinces, although it is an interesting coincidence at least that the first official records from all these areas should all be in the same year. It is much more probable that dwarf bunt was in existence for a matter of years in each area before being officially recognized. Perhaps Tingey's (1950) circumstantial evidence that the disease was established in Utah in the 1920's, or even perhaps by 1903, can be officially substantiated by study of old collections of wheat bunt from the area.

In this connection, we are hoping that our colleagues in other states (that is, other than New York and Washington) and countries where dwarf bunt occurs or logically could occur will restudy herbarium specimens and other reliable collections from their own regions in a cooperative effort to more definitely determine as accurately as possible when and where this important disease was first found. Most such specimens and collections will contain only the smutted heads of wheat and thus will give little clue as to any dwarfing effect. However, there are other diagnostic characters that are useful: sori (smut balls) more round and compact, and tending to expand the lemma and palea more than in T. caries; reticulations (areolae) large and deep (mostly 3 to 5 μ wide and 1.5 to 2.5 μ deep); spores individually encased in a hyaline sheath 1 to 4 μ thick. It is recommended that the microscopic examinations be made with an oil immersion lens and that the spores be mounted in Shear's mounting fluid and heated slightly (Fischer, 1952).

All collections identified as Tilletia tritici (Bjerk.) Wint. or T. caries (DC.) Tul. should be restudied. We should be very glad to assist in the diagnosis of any questionable collections. These may be sent to either author.

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WASHINGTON AGRICULTURAL EXPERIMENT STATIONS, PULLMAN, WASHINGTON, AND CORNELL UNIVERSITY AGRICULTURAL EXPERIMENT STATION, ITHACA, NEW YORK

KERNEL BLIGHT PHASE OF SEPTORIA BLACK STEM OF OATS

M. D. Simons and H. C. Murphy

The leaf spotting phase of Septoria black stem (Leptosphaeria avenaria Weber) (Septoria leaf blotch or speckled blotch) was described by Weber (4), and the black stem phase by Meehan and Murphy (1) and Poole and Murphy (2). As reported by Stanton (3) the kernel blight phase of Septoria black stem of oats was widespread throughout most of the North-Central States during 1952. The authors found that the causal organism could be isolated from blighted kernels that had been in storage for over a month. This raised the question of whether the disease could be transmitted through the seed.

Seed of Cherokee oats was divided into five classes, based on severity of symptoms, as follows:

1. Groats heavily blackened and shriveled.
2. Groats moderately blackened, but not shriveled.
3. Groats lightly blackened.
4. Lemmas blackened, but groats showing no evidence of infection.
5. Check. Neither groats nor hulls showing evidence of infection.

All seed was hulled and sown in sterilized sand in the greenhouse in four replications. Seedlings were examined nine days later.

The emergence percentage of the heavily blackened, shriveled groats was significantly lower than that of the other classes. There were no differences between the emergence percentages of the other classes. None of the seedlings of any of the classes showed evidence of infection. The lower emergence percentage of the heavily blackened and shriveled kernels apparently resulted mainly from the damage caused by the fungus prior to harvest. All plants were grown to maturity without expressing any of the symptoms normally associated with the disease.

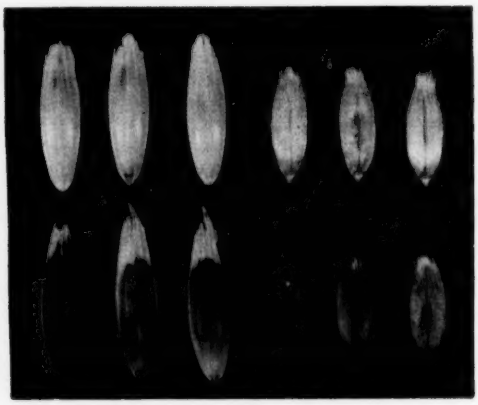

FIGURE 1. Cherokee oats. Above -- Non-infected with Septoria. Below -- Lemmas and caryopses showing symptoms of Septoria black stem infection.

In order to determine whether the fungus was able to attack seedlings at the lower temperatures ordinarily prevailing during germination of oats under field conditions, different lots of seed were grown in controlled temperature chambers at 10°, 15°, and 20° and in the greenhouse at temperatures of 25° C., and above. Three classes of seed were grown in sterile sand at each temperature. These classes were as follows:

1. Groats heavily blackened and shriveled.
2. Lemmas heavily blackened, but seed not hulled.
3. Check. Lemmas showing no blackening, seed not hulled.

There was no evidence of seedling infection at any of the temperatures. Consequently, it would appear that the Septoria black stem disease is not transmitted through infected seed.

The possibility that Septoria-blighted grain might be toxic to livestock was also considered. The authors are indebted to Dr. S. G. Kanzy and Mr. John Mathews, Jr., of the staff of the Veterinary Research Institute, Iowa State College, for carrying out feeding tests using mice and chickens. Two lots of oats were used for the feeding tests. One was a lot of Cherokee grown near Cresco, Iowa, in 1952. Approximately 5 percent of the Cherokee kernels showed symptoms of Septoria infection, ranging from slightly darkened lemmas to completely black and shriveled kernels. The other lot of seed was Septoria-free Cherokee grown near Aberdeen, Idaho, in 1951 (Figure 1). Dr. Kanzy and Mr. Mathews found no evidence of toxicity of Septoria-infected grain to either mice or chickens under the conditions of the experiment. It should be emphasized, however, that tests were not conducted with any large domesticated animals, and consequently their

reaction to the blighted grain cannot be reported.

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COOPERATIVE INVESTIGATIONS BETWEEN THE DIVISION OF CEREAL CROPS AND DISEASES, U. S. BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AND THE IOWA AGRICULTURAL EXPERIMENT STATION.

CERTAIN FACTORS AFFECTING CHLOROPHYLL DEGENERATION AND PIGMENTATIONS IN OATS AND OTHER GRASSES

H. H. McKinney, A. W. Specht, and T. R. Stanton[1]

INTRODUCTION

A red-leaf condition in oat fields in the eastern half of the United States has attracted considerable attention since 1949. Some workers regard the trouble as a wholly new disorder. Many factors are responsible for the red colorations that sometimes appear in oats and many other plants, and for this reason, as Sprague (10) has pointed out, "It is not necessarily a symptom of some specific disease ....."

Leaf reddening in oats was conspicuous in the central and eastern spring-oat region in 1907, 1908, and 1909, and there was much speculation at that time regarding the cause (4). In 1936 Sprague (9) discussed reddening in oats and its various causes in western Oregon. He stressed low temperature, root and crown rotting fungi, poor soil drainage, and low nitrogen supply as being among the important causal factors operating in areas that had come under his observation. In 1937 Barrus (1) called attention to leaf reddening in oats in New York State and stated that the trouble had been observed there for many years. He reported that blasted panicles were more frequent among reddened than among non-reddened plants.

GENERAL OBSERVATIONS

In 1950 the senior author discussed pigment disorders in winter small grains in the Southeastern States (6). Since that time many observations have been made in the spring-oat areas, and on several of the summer annual grasses that frequently develop anthocyanin in the leaf blades, sheaths, and exposed stems. During the past three years many samples of oats labeled "red-leaf" have been received. Also, workers and growers have called attention to "red-leaf" in oats in experimental plots as well as in commercial fields. In the majority of these cases the oat foliage was actually not red, but chiefly shades and tints of orange, such as cinnamon-rufous and vinaceous tawny; red hues of orange, as rufous and terra cotta; and yellow hues of orange, as yellow-ochre.[2]

The several plant pigments are dealt with in a general way only in this report. Many text and reference works are available from which detailed information may be obtained. Two reference works on the anthocyanin pigments, Onslow (7) and Blank (3), are especially helpful.

Microscopic examinations of free-hand sections of oat leaves have shown that the many shades and tints of red, ranging from maroon and ox-blood through to carmine, scarlet, and violet hues, are due chiefly to the anthocyanins located in the epidermal cells on either or both sides of the leaf. On the other hand, the orange, reddish orange, or terra cotta and yellowish orange pigments are located in the mesophyll tissue, and these pigments are unmasked with the depletion of the chlorophyll when the epidermal cells are free or nearly free of anthocyanin. These pigments consist of carotenoids and yellow or terra-cotta water-soluble pigments. Traces of anthocyanin and possibly red degradation products of the carotenoids may contribute to the terra-cotta colorations in the mesophyll. Sometimes the chlorotic leaves of oats are yellow, but yellow has been observed less frequently in oat leaves than in the leaves of wheat and barley. Traces of red anthocyanin in the epidermal cells sometimes contribute to the reddish copper color in the chlorotic leaves. Brownish colorations in the walls of the epidermal cells over the several subepidermal colors seem to explain the tan (yellow-ochre) colorations. The color of these walls also modifies the visual appearance of the anthocyanins and the pigments in the mesophyll.

Many observations in different localities indicate that the premature expression of these copigmentations in the mesophyll, and of the bright red (carmine and scarlet) anthocyanin in the epidermis, essentially represent early senility following premature depletion of the chlorophyll. Under favorable conditions individual leaves of healthy, adapted oat varieties should retain most

[1]The authors wish to acknowledge the assistance of members of the Division of Soil Management and Irrigation Agriculture: Myron S. Anderson for many helpful suggestions, V. J. Kilmer in making the pH determinations, and E. M. Roller in making nitrogen determinations; and the cooperation of F. A. Coffman of the Division of Cereal Crops and Diseases for access to his nurseries for study, and for supplying seed.

[2]The names of colors used in this paper are from R. Ridgway's, Color Standards and Color Nomenclature. 43 pp., illus. Washington, D. C. 1912.

of their chlorophyll for about five to six weeks or more after the leaf tips emerge from the sheaths in either autumn or spring. The normal functions of the leaf are impaired when the amount of chlorophyll that is characteristic of a variety becomes greatly reduced or depleted within an appreciably shorter period. This is detrimental to the plant, irrespective of the final color of the carotenoids, of the presence of anthocyanins or anthoxanthins, or of the parasitic or non-parasitic causes of chlorophyll depletion.

It has been observed, further, that there are two extreme manifestations in the behavior of the plastid pigments in the leaves of non-parasitized grasses during senility and death. In one, depletion of the chlorophyll in the leaf is comparatively slow, and depletion of the remaining pigments, complete loss of turgidity, and death of the tissue, all follow chlorosis in very close succession, beginning at the tip of the leaf. In the second manifestation, the chlorophyll depletion is rapid, leaving the other pigments exposed over a large portion or the whole of the leaf. These chlorotic leaves may remain turgid for several days before the pigments decompose, and the tissue dies. The discolored leaves are very conspicuous when they are near the top of the plant. Frequently the yellow and terra-cotta pigments decompose slowly in this dead tissue, as they are plainly visible in the mesophyll cells of the moistened tissue when viewed through a microscope.

Up to two or three weeks before maturity, a field of healthy, vigorous oats has a canopy of green leaves that obscures the senile and dead basal leaves on normal plants from view. Oat yields usually are impaired when the chlorotic leaves and/or the bright red leaves are readily evident amid the remaining green foliage of immature plants.

Plastid pigment disorders, and the expression of the red, carmine, and scarlet anthocyanin observed on oats, U. S. Hybrid 13 field corn, crabgrass, and other wild grasses occurred mostly on land of medium or low fertility and in poor physical condition. Some of the premature orange colorations have been observed in oats growing in fertile fields, but usually these plants were injured by aphids, root and crown rotting fungi, or leaf spotting fungi. Aphids and fungi are sometimes found associated with these pigment disorders on poor land. In fields of reddened oats it was observed that the amount of fertilizer applied is sometimes too scant, as indicated by plants that were green and thriving in spots where fertilizer was spilled when the distributing machine was filled from the supply truck.

From the observations made in these studies, it appears that the expressions of maroon, oxblood, and violet tinted anthocyanin are not especially serious danger signals in oats, since leaves so affected usually have shown little if any impairment of the chlorophyll. Serious damage is indicated by premature chlorosis, regardless of other colors.

The observations are in accord with the literature in that they indicate that the plant functions which respond to the meterological and edaphic factors and regulate the color of chlorotic mesophyll tissue, are in part independent of those which regulate the color of the epidermal tissue.

EXPERIMENTAL

Early in the experimental work it was found that the expression of anthocyanins in oats tended to be erratic. For this reason, attention has been given to certain wild grasses, corn, and a few non-grass species. Smooth crabgrass, Digitaria ischaemum (Schreb.) Muhl., hairy crabgrass, D. sanguinalis (L.) Scop., broomsedge, Andropogon virginicus L., have been helpful, but these species, like oats, have strains that differ in their ability to produce anthocyanin pigments. U.S. Hybrid 13 field corn, a purple field corn (C. I. 7 x Kys) x (ABPl), red cabbage, and red-leaved barberry, Berberis thunbergii DC., also were studied.

Light and Temperature

Direct, unobstructed sunlight has favored the expression of the anthocyanin pigments in all the plants listed, including oats. The interception of sunlight by ordinary window glass, which is an effective filter against the short rays of the spectrum, the ultra violet in particular, delayed, reduced, or prevented the expression of these pigments in the grasses and cabbage. Barberry was not tested, and oats did not always express anthocyanins when expected. Cool temperatures likewise favored the expression of anthocyanin in the grasses, but cabbage and barberry were not tested. The crabgrasses express anthocyanin colorations at mid-summer temperatures out-of-doors. However, in concurrent tests conducted during the autumn it was observed that cool night temperatures accentuated the expression of anthocyanin, whereas, warm night temperatures near 80° F. prevented the expression of this pigment, even though the plants in both tests received full unobstructed sunlight and experienced the same temperatures from sunrise to sunset.

In the field the expression of the anthocyanin pigments in oats and in many other species be-

comes most evident in plants at the borders of fields or blocks, and in spaced plants where exposure to the direct rays of the sun is greatest. Furthermore, these pigments frequently come to expression in the field following a period of cool temperatures. All of these observations on sunlight and cool temperatures are in accord with the observations of many of the early ecologists, particularly those who studied alpine vegetation (7).

Anthocyanin appears first in the leaf epidermis that receives the direct rays of the sun, in certain varieties of oats, U. S. Hybrid 13 field corn, crabgrass, and barberry, but it appears eventually in the epidermal cells of both leaf surfaces. Microscopic examinations of these plants revealed little or no chlorophyll in mesophyll tissue in parts of leaves having crimson to scarlet anthocyanin in both epidermal layers.

The sheaths and the exposed stem tissues of all the plants of the crabgrasses and the U. S. 13 field corn expressed some degree of anthocyanin coloration when cultured under any of the conditions of these tests, although the expression of anthocyanin in these parts was weakened under conditions favoring anthocyanin-free leaf blades. On aging, however, the "anthocyanin-free" blades of U. S. 13 corn have always shown red anthocyanin along the edges, extending inward from eight to ten cells.

In leaves of purple field corn grown at 70° to 75° F., in a greenhouse during the winter, the red anthocyanin came to mild expression in the mid-ribs, in small dots in the basal region of the leaf blades, and in small streaks over the large vascular elements. This red pigment seemed to be confined to epidermal cells. At 50° the blades contained much purple (blue and red) anthocyanin. Although anthocyanin was clearly evident in the sheaths, husks, and stems at the higher temperature, it was much stronger at 50°, though not nearly so strong as in the field.

In field culture of this corn the blue and red anthocyanins appear to be present in the parenchyma as well as in the epidermal cells of the leaves. Blue anthocyanin was also observed in immature seeds shielded from the sun by all of the immature purple to red husks. Even in the field, however, anthocyanin is less intense to the eye in the leaf blades than in the sheaths, husks, and exposed stems, and it is less intense in shaded tissues than in tissues receiving direct sunlight.

Microscopic examinations of the purple corn leaves, and all general observations in the field, indicate that the blue and red anthocyanins do not forewarn or indicate chlorophyll degeneration in this corn.

Effect of Mineral Nutrition on Leaf Discoloration

Plastid pigment disorders and anthocyanins had been observed so frequently in some of the grasses growing on poor land that considerable attention has been given to the factor of mineral nutrition. In tests with crabgrass, amply watered plants growing singly in soil in clay pots produced more anthocyanin in three-inch than in six-inch pots. This observation strongly suggested that the expression of anthocyanin is favored by the depletion of mineral nutrients.

Tests with a complete nutrient solution, including the minor elements, quickly demonstrated that the expression of anthocyanin can be prevented in the leaf blades and be greatly reduced in the sheaths and exposed stems of crabgrass when the nutrient level is kept sufficiently high. The expression of anthocyanins in certain varieties of many plant species has been regarded as an indicator of deficiencies of nitrogen or phosphorus (2). However, it was suspected that nitrogen was the critical element in the soils under test, and the use of a 1/6 percent solution of ammonium nitrate gave the same results as did the full nutrient solution. Leaf blades of crabgrass plants out-of-doors with unobstructed sunlight showed no anthocyanin when the plants received sufficient nitrogen. Plants in the same situation that received no nitrogen had anthocyanin in all except the youngest non-shaded leaves. The maroon, ox-blood, and violet tinted anthocyanin has been observed to disappear from a leaf after an application of nitrogen to the soil.

Pot tests with oats were conducted out-of-doors on several occasions with soils of the Branchville Series and the Keyport Series, both high in clay and low in organic matter. Oats on both soils in the field had manifested chlorophyll depletion and in some cases reddening of the leaves the previous season. The reaction of these soils is near pH 7.2. The spring varieties Cherokee, Marion, and Kanota, and the winter varieties Arlington, Traveler, and C. I. 5911 (N. J. A-2-2), were used in the tests.

In several preliminary tests it was observed that applications of a commercial fertilizer, a complete nutrient solution including the minor elements, calcium nitrate alone, and ammonium nitrate alone, in sufficient amounts, prevented premature degeneration of the chlorophyll, providing root and crown rotting parasites were not operating. When the roots and crowns were infected many of the chlorotic leaves were striped or solid with shades of orange or terra cotta, usually with no signs of anthocyanin. Unmistakable lesions and the death of the plants or tillers enabled

quick diagnosis of infection. Even in infected plants, however, chlorophyll degeneration was slower when nutrition was ample. An occasional leaf on healthy plants developed ox-blood or pomegranate purple anthocyanin after a cool period when the nutrition level was high, but there was no evidence of serious chlorophyll depletion associated with these anthocyanins.

All of the premature chlorophyll degeneration and the development of anthocyanins which sometimes is associated with this degeneration in non-infected plants, occurred in the plants that received insufficient or no additional nutrients. As with crabgrass, oats showed the importance of adequate amounts of nitrogen for the reduction and prevention of premature depletion of the chlorophyll, in the soils used in the tests.

When these soils were steamed, oat plants grown in them had the same dark green color and vigor as plants growing in the non-steamed soil to which nitrogen was applied. As there were no signs of infection in the roots or crowns in any of these plants, nor in the control plants growing in the native field soil, it is concluded that the beneficial effects from steaming were due largely to an increase in the soluble nitrogen which usually attends sterilization in many soils.

A combined yield and nutrition test with spring oats is discussed in detail. The soil was the Branchville Series. Cherokee and Kanota oats were used. Two six-inch clay pots with free drainage were used for each soil treatment for each oat variety. Three plants were grown in each pot. The fertilizer was thoroughly mixed with the soil just before seeding. The nitrogen treatment was made with a 1/3 percent solution of calcium nitrate applied in four equal portions to make the total shown in Table 1, beginning with seeding and ending five days before the panicles of Cherokee began to lose chlorophyll and ripen.

The chlorophyll ratings in Table 1 are averages of the comparative visual intensity of the green color of each leaf. Seven classes, ranging from 0 for no chlorophyll, to 6 for the most intense green, were recorded. Fractional values were used when parts of leaves were free of chlorophyll. The final averages for each date in each of the sets are based on the number of green leaves per primary culm of the plants getting nitrogen. The primary flag leaves were completely extruded in Kanota on June 4, and Cherokee in the control set was within five days of ripening on June 25 when the panicles were beginning to lose their chlorophyll.

Based on the Ridgway color standards the most intense green (the nitrogen set) of Cherokee oats matched Varley's green. The green of next intensity (high fertilizer set) was between Varley's green and Civette green. The weakest green was in the control and the lowest fertilizer set, and matched chromium green. In the Kanota variety, the intensity of the green was just slightly less, and the rate of chlorophyll degeneration was slower than in Cherokee.

In plants getting the calcium nitrate or the highest application of fertilizer, the chlorophyll did not show signs of appreciable degeneration until five to six weeks from the time of the emergence of the leaf tips from the sheaths. With the reduced amounts of fertilizer, this time gradually shortened to three weeks or less in the control plants.

Cherokee and Kanota produced seven and eight leaves, respectively, per primary culm, and Cherokee was one week earlier than Kanota. In both varieties, the chlorotic leaves were tones of orange and yellow-ochre, with a tendency towards yellow in Kanota. Both varieties, especially Cherokee, produce anthocyanins, but they were erratic in this particular, apparently partly on account of the lack of sufficiently low temperatures. Following a cool period, ox-blood anthocyanin appeared in leaves 5 and 6 of Cherokee plants that received nitrogen only, but with no signs of damage to the chlorophyll. In the remaining sets, Cherokee showed traces of violet hue of red (spinel red and pomegranate purple) in leaves 5 and 6, whereas in Kanota, traces of this red appeared in leaf 6 in the control set only.

The highest yields of grain (Table 1) of both Cherokee and Kanota oats were from the 4-12-4 fertilizer application at the exorbitant rate of 6385 pounds per acre (255 lbs. of nitrogen per acre). The second highest yield of Kanota resulted from the calcium nitrate application at the rate of 120 lbs. of nitrogen per acre. The Cherokee grain yields were about the same from the nitrate and from 4788 lbs. of fertilizer per acre. The mixed fertilizer was applied at the beginning of the test, whereas the calcium nitrate was applied in four equal portions, at intervals from seeding to panicle development. The strongest expression of green color was in the nitrate treatment, but the highest grain yields were from plants having a somewhat weaker green color but no premature depletion of the chlorophyll.

The heavy applications of fertilizer and nitrogen that induced the best results are far in excess of those recommended in field practice. However, to demonstrate the physiological manifestations under investigation, it is clear that for the soil used, in pot culture, very large quantities of nutrients were necessary. When clay pots with free drainage are located out-of-doors, leaching of the soluble salts is greatly in excess of that in a field of silt loam soil over a tight sub-soil. During the period of the pot tests, there was much rain which had a marked leaching effect, and all of the leachings were lost to the plants. Just before harvesting the oats in the pot

Table 1. Influence of nutrition on chlorophyll and yield of Cherokee and Kanota spring oats grown in pots out-of-doors. Seeded April 11, 1951. Harvested Cherokee July 12, and Kanota July 20.

Plant characteristics	Soil treatments											
	No treatment (Control)		Commercial fertilizer (4-12-4) grade								Calcium nitrate	
			532 lbs.[a]	1 gram[b]	1596 lbs.	3 grams	4788 lbs.	9 grams	6384 lbs.	12 grams	710 lbs.	1.333 grams
	Cherokee	Kanota	Cherokee	Kanota	Cherokee	Kanota	Cherokee	Kanota	Cherokee	Kanota	Cherokee	Kanota
Number of fertile heads	14.0	8.0	10.0	10.0	14.0	14.0	21.0	30.0	25.0	33.0	30.0	36.0
Weight of grain (gm.)	7.2	5.8	10.0	7.7	14.1	14.1	27.7	27.5	31.0	32.6	27.5	29.0
Weight of straw and chaff (gm.)	9.8	7.1	11.3	11.1	17.1	18.8	29.3	37.8	38.4	44.8	32.6	30.8
Total dry weight (gm.)	17.0	12.9	21.3	18.8	31.2	32.9	57.0	65.3	69.4	77.4	60.1	59.8
Chlorophyll ratings												
June 4	1.7	2.9	1.8	2.9	2.8	3.0	2.9	3.0	4.5	3.8	5.3	4.7
July 25	1.5	2.8	2.0	2.5	2.1	2.7	2.5	3.1	2.9	4.1	5.6	5.2

[a] Rate of application in pounds per acre
[b] Rate in grams applied to each pot
(Calculations are made on an area basis)

test, the soil in each of the treatments, including the control, was sampled for the determination of nitrogen. It was found that the nitrogen content in all of the soils was almost the same as in the no-treatment control, which contained 0.106 percent nitrogen.

The effect of drought was observed in a pot test. Green oat leaves that were within five to seven days of the end of their normal functioning period lost their chlorophyll very quickly and became orange in color when about one-half of the water required to keep the soil well moistened at all times was suddenly withheld for two hot days. This was particularly striking in the high-nitrogen plants because of the contrast with the remaining dark-green leaves.

The present studies, and studies of oat nurseries, seem to give reason for the belief that many varieties and lines, among them being Marion, Cherokee, C. I. 5911, C. I. 5936, and Traveler, have rather exacting requirements for the proper maintenance of their chlorophyll. Studies made thus far suggest that Arlington oats may be less exacting, but nevertheless more so than are many varieties of wheat. In general, chlorophyll seems to be less stable in oats than in wheat.

Effect of Soil Reaction

Most of the world oat crop is grown on soils having a pH below 7.2, the reaction of the soil used in the above experiments. Since ample amounts of nitrogen had prevented premature degeneration of the chlorophyll, and induced a good yield of grain in oats grown in this soil, it was decided to make a further study of the effects of nitrogen when the soil is adjusted to a range of pH reactions. Hydrated lime or flowers of sulfur was added to the field soil under study (Branchville Series). The soils were then kept moist in glazed crocks 4 inches in diameter and 2.5 pints capacity, and stored in a greenhouse from January to October, and out-of-doors exposed to the weather from October to April of the following year. In April the soils were worked up and their reactions determined, and Cherokee and Kanota oats were planted. The reactions ranged from pH 3.8 to pH 7.8. The buffering capacity of the soil was reached at each end of the series.

Three oat plants were grown in each crock, and four crocks were devoted to each treatment for each oat variety. The crocks were spaced for uniform exposure to direct, unobstructed sunlight. As soon as the seedlings started to emerge, 50 cc. of a 1/6 percent solution of ammonium nitrate was applied to each crock, except in the sets to which no lime or sulfur was added. These applications were continued at suitable intervals until a total of 200 cc. of nitrate solution per crock had been applied. At the fourth and final application, panicles of Cherokee were beginning to extrude. Panicles of Kanota appeared five days later. The rate per acre of nitrogen applied was very close to that of the calcium nitrate test (Table 1), about 120 lbs. per acre.

The oat plants were killed in the seedling stage in soil at pH 3.8. At pH 4.0 they were less thrifty than at higher pH levels, and gave the lowest yields of grain.

The intensity of the green color and the stability of the chlorophyll in the leaves were rated from 1 to 4 throughout the season. The rating 1 indicates the weakest green and the greatest amount of leaf tissue in which the chlorophyll was depleted prematurely. The rating 4 indicates a strong green color, with little or no premature depletion of chlorophyll.

The highest seasonal average chlorophyll rating, 3.6, was obtained from plants at pH 4.9, 6.0, and 6.7. The next highest rating, 3.2, was obtained at pH 7.2, 7.4, 7.8, and 7.8, the latter representing an excess of lime. At pH 4.0, the chlorophyll rating was 2.0. The control, which received no nitrogen (pH 7.2), had a chlorophyll rating of 1.6.

In both varieties, the highest average grain yields were from the sets grown at pH 7.2 and above, and the highest yields in the test were from plants in the soil containing the greatest amount of lime, pH 7.8. As in the test cited in Table 1, the plants having the strongest green color did not produce the highest yield of grain. There was no evidence of premature degeneration of chlorophyll in the leaves of plants growing at pH 4.9 and above, when the nitrogen supply was ample.

Early in the season a few leaves in the lime series showed traces of purple anthocyanin, but this was of no consequence. Red anthocyanin appeared in a few scattered leaves of Kanota during mid-season, but this was of no apparent consequence, as the affected leaf tissues were nearing the end of a normal functioning period.

Effects of Aphids on Leaf Discoloration[3]

A yellow aphid, Sipha flava (Forbes), is found at Beltsville, Maryland, associated with red

[3]Identifications of the aphids were made by Louise M. Russell through the cooperation of F. W. Poos, Bureau of Entomology and Plant Quarantine.

leaves of the crabgrasses Digitaria sanguinalis and D. ischaemum, and of Paspalum laeve Michx. and P. stramineum Nash. This aphid is very common on grasses in the Southern States, and in certain areas of the tropics. It was found in great abundance on crabgrass cattle pastures in Puerto Rico in 1952. Louise Russell, who made the observations in Puerto Rico, reported that the leaves of the crabgrass in the aphid-infested pastures showed much reddening.

Feeding tests at Beltsville have shown that this aphid induces anthocyanin in the epidermal cells of the grasses listed. The crabgrasses received the most attention. Leaves of plants grown out-of-doors and in the greenhouse developed maroon to red anthocyanin after one day of aphid feeding. Frequently entire leaves reddened and degenerated. The leaf sheaths frequently reddened a distance of 2 or 3 cm. below the ligule. The anthocyanin spread from the feeding spots more rapidly in plants receiving no supplemental nitrogen than it did in those receiving ample nitrogen. Also, the anthocyanin developed the bright red colors more readily out-of-doors in bright sunlight than under glass. This aphid has induced anthocyanin in the leaves of very small seedlings as well as of older plants. Depletion of the chlorophyll in the leaves was hastened in the area involved by this aphid-induced anthocyanin. When the aphid-infested plants were cultured under conditions not favorable for the expression of anthocyanin, the leaves became spotted or solid yellow prematurely, with diffuse brownish zones surrounding the feeding punctures.

Greenhouse-grown plants on which Sipha flava did not feed, and which served as controls, showed no reddening nor premature chlorosis in any of their leaves. Controls in tests conducted out-of-doors developed less reddening than the aphid-infested plants.

The aphid is rarely found on very young leaves, and it thrives in hot, sunny situations. It usually feeds and colonizes on the underside of the leaves. Under heavy infestation the insects will feed on the sheaths and stems. In nature it feeds on many crabgrass plants on which it does not colonize or leave a visible injury at the points of feeding. This makes it difficult to correlate the red leaves with the insect in many cases. Such reddened leaves cannot be distinguished from those in which the reddening is induced by malnutrition or other non-parasitic factors that induce the expression of anthocyanin.

Crabgrass seedlings with red leaves from one day's feeding by this aphid have been kept under close observation in a greenhouse until maturity, and there were no signs of systemic reactions in any of the plants. The entire reaction induced by the aphid in the crabgrasses and in the species of Paspalum is local, as with the mosaic-like mottling in peanuts caused by an unidentified aphid in Sierra Leone, West Africa (5), and with the localized chlorosis and reddening in alfalfa and several other plants induced by several phloem-feeding leaf hoppers (8). Furthermore, manual inoculations of healthy leaves with the juice from reddened leaves have failed to give any sign of transmission of a virus that remains local. Until improved methods may demonstrate otherwise, the most tenable working theory is that these insect-induced pigment disturbances are of non-virus nature.

Summer-grown oats (C. I. 5936), are not so congenial as a host to this aphid as is crabgrass, but the insect will feed and colonize on the leaves. Chlorophyll disappears from the insect feeding areas in oats in less than 24 hours when the nitrogen level is low, leaving orange colored chlorosis visible on both sides of the leaf. When the nitrogen level is high, appearance of chlorosis in the feeding areas is delayed. After a 56 hours' feeding period on high nitrogen plants, the feeding areas had a dark water-soaked appearance, and the chlorophyll showed signs of depletion in these areas from 24 to 36 hours later. The spread of chlorosis from the feeding areas was relatively slight. On low-nitrogen plants, entire leaves lost their chlorophyll when the aphid infestation was moderately heavy, leaving the orange or terra-cotta pigments exposed. Sipha flava colonized more readily on the low than on the high-nitrogen plants.

In the spring of 1950, several lines of oats in the nursery at Beltsville developed orange and terra-cotta colorations, following an early infestation of unidentified aphids. As the season advanced, it was observed that many of these lines showed marked recuperation. The new foliage contained chlorophyll and the plants matured, though somewhat stunted. The reactions in these oats did not suggest a virus infection.

During the winter of 1950-51, the apple-grain aphid Rhopalosiphum prunifoliae (Fitch) was present in the oat greenhouses, which were kept near 55° F. at night, and near 65° during the day. It was observed that entire leaves in some of the lines of oats, particularly in C. I. 5936, Landhafer X (Mindo x Hajera-Joanette), on which this aphid colonized, became chlorotic, the exposed pigments in the mesophyll tissue being colored in shades of orange and terra-cotta, as in the field. After the greenhouses were fumigated with Nicofume, and most of the aphids were killed, it was observed that the new leaves came out green and continued so if they did not become reinfested with aphids. This problem requires further study. However, under the conditions of the observation, this aphid-oat reaction strongly suggests a direct effect of aphid feeding rather than a virus

infection, resembling in principle the aphid-induced anthocyanin reaction and chlorophyll depletion in crabgrasses and Paspalum spp.

Anthocyanin Spots Caused by Acid

Crabgrass plants growing in the vicinity of a coal-burning heating plant frequently developed maroon to ox-blood anthocyanin spots on the leaves. These spots had a small necrotic central spot suggesting insect feeding punctures, but closer observations revealed that the spots were associated with small cinders immersed in dew drops or rain drops on the leaves. As would be expected, these cinders gave an acid reaction in water. Direct applications of very dilute sulfuric acid likewise induces anthocyanin spots in crabgrass leaves receiving unobstructed sunlight.

DISCUSSION AND SUMMARY

The effects of light, temperatures, mineral nutrition, soil reaction, aphid infestation, and acid burning on chlorophyll degeneration and the development of anthocyanin pigments in oats and other grasses was investigated.

Nitrogen seems to be the chief element in short supply in the soils used in these tests. Premature degeneration of the chlorophyll in oats was prevented when mineral nutrients, particularly nitrogen, were supplied in sufficient amounts and plant parasites and drought injury were not present. The expression of anthocyanin pigments is more erratic in oats than in certain of the wild grasses such as the crabgrasses. Adequate supplies of nitrogen reduced the red (carmine to scarlet) anthocyanins that sometimes attended premature degeneration of chlorophyll in oats. It appears that the very dark shades of anthocyanin (maroon to ox-blood and tints of purple and violet) sometimes associated with the high-nitrogen oat leaves may be of little or no value as a sign of serious trouble in oats.

It is suspected that adequate amounts of fertilizer, particularly nitrogen, will do much towards increasing the stability of chlorophyll in oats in the Southeastern States where unexplained cases of chlorosis and anthocyanin pigmentation are frequently observed. Additional top-dressings may be needed where rains have leached much of the nitrogen and other soluble salts.

In our pot tests, the very heavy application of nitrogen that induced the maximum green color in the oat leaves resulted in smaller yields of grain than more moderate applications in the complete fertilizer. There was no evidence of premature degeneration of the chlorophyll in the leaves of the high yielding plants, however.

An evaluation of oat varieties and lines for stability of their chlorophyll systems, as influenced by nutrition, temperature, sunlight, and soil moisture, should aid in the diagnosis of chlorophyll disturbances caused by viruses and other parasites, and also facilitate oat breeding. Without improved methods for diagnosis, much of the selection work will be haphazard, and oat improvement will be retarded.

Photographic records in color (35 mm. size), were made of most of the typical materials from the tests reported in this paper. Copies of these are available at cost through the American Type Culture Collection, 2029 M Street, N. W., Washington 6, D. C.

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A NEW STRAIN OF TOBACCO RINGSPOT VIRUS ISOLATED FROM BEAN

Pen Ching Cheo and W. J. Zaumeyer[1]

In the fall of 1951 severely mottled pods of Bountiful bean, Phaseolus vulgaris L., were collected in Virginia. Inoculations with juice of such pods to tobacco, Nicotiana tabacum L., cowpea, Vigna sinensis (Torner) Savi, and several bean varieties demonstrated that the disease was of virus nature, and the symptoms produced on tobacco (Fig. 1, M) resembled those produced by the tobacco ringspot virus. The same virus had been isolated from beans in another collection made in Delaware several years ago. No field survey has been made for this virus, but the severe symptoms it produces on most bean varieties would make it of economic importance if it is widespread.

This paper deals with the identification of this virus, describes the symptoms of the disease on certain hosts in the greenhouse, and compares the virus with three other strains of tobacco ringspot virus.

MATERIALS AND METHODS

All the inoculations were made under greenhouse conditions at Beltsville, Maryland. About ten plants of each bean variety and of each of the other species were used for each inoculation test. Juice of infected Idaho Pinto or Topcrop variety was used as the source of inoculum in all inoculations. In order to compare the new virus with related strains the following strains of tobacco ringspot virus were used: 1, McKinney's strain of tobacco ringspot virus; 2, Pound's (5) yellow strain of tobacco ringspot virus isolated from watermelon; and 3, Fulton's green strain of tobacco ringspot virus. The last two strains were supplied by G. S. Pound of the University of Wisconsin.

EXPERIMENTAL RESULTS

Reactions of Bean Varieties

Every variety of bean tested was susceptible to the virus. The reaction was almost wholly necrotic (Fig. 1, A-F). Local symptoms usually consisted of chlorotic or both chlorotic and necrotic flecks, spots or ringspots with occasional streaking of the veins (Fig. 1, F) and epinasty of the primary leaves. In some cases streaking of veins (Fig. 1, B) and leaf epinasty were the only local symptoms produced. The local necrosis often became so extensive and severe that rapid killing of the inoculated leaves resulted. The systemic symptoms that usually followed consisted of necrotic spots on the trifoliate leaves and pulvini, petiole and top necrosis, and finally death of the entire plant. The extent of such expression depended on the susceptibility of each variety. The results of the tests are shown in Table 1.

In general, the bean virus strain incited more severe symptoms on most bean varieties than did the McKinney strain of tobacco ringspot virus; exceptions were Idaho Refugee, Michigan Dark Red Kidney, and Rival varieties, which were more severely infected by the McKinney strain. The symptoms produced by the two viruses showed characteristic differences. On most varieties (Table 1) the bean strain produced local chlorotic areas, spots or rings, which were sometimes concentric (Fig. 1, A). These chlorotic spots or rings later became necrotic, or only the borders of the lesions were necrotic (Fig. 1, D). Sometimes they remained chlorotic (Fig. 1, C). In certain cases only a very minute necrotic dot was produced at the center of each chlorotic spot. This chlorotic local symptom was never observed on any bean variety inoculated with the McKinney strain. Moreover, epinasty of the primary leaves appeared to be a typical symptom on most bean varieties inoculated with the bean strain (Fig. 1, H); when inoculated with the McKinney strain only a few varieties showed this symptom (Table 1). It appears that (Table 1) the yellow strain of the tobacco ringspot virus is closely related to the McKinney strain, while the green strain is closely related to the new bean strain. The yellow strain appears to be the most severe of the four virus strains and the green strain the mildest.

Reactions of Other Plants

Table 2 records the symptoms produced by the four strains on some selected hosts. In general, the bean strain produced a milder effect than the McKinney strain on most of the hosts

[1]The services of the senior author were available for this work through a grant of the U. S. Department of State.

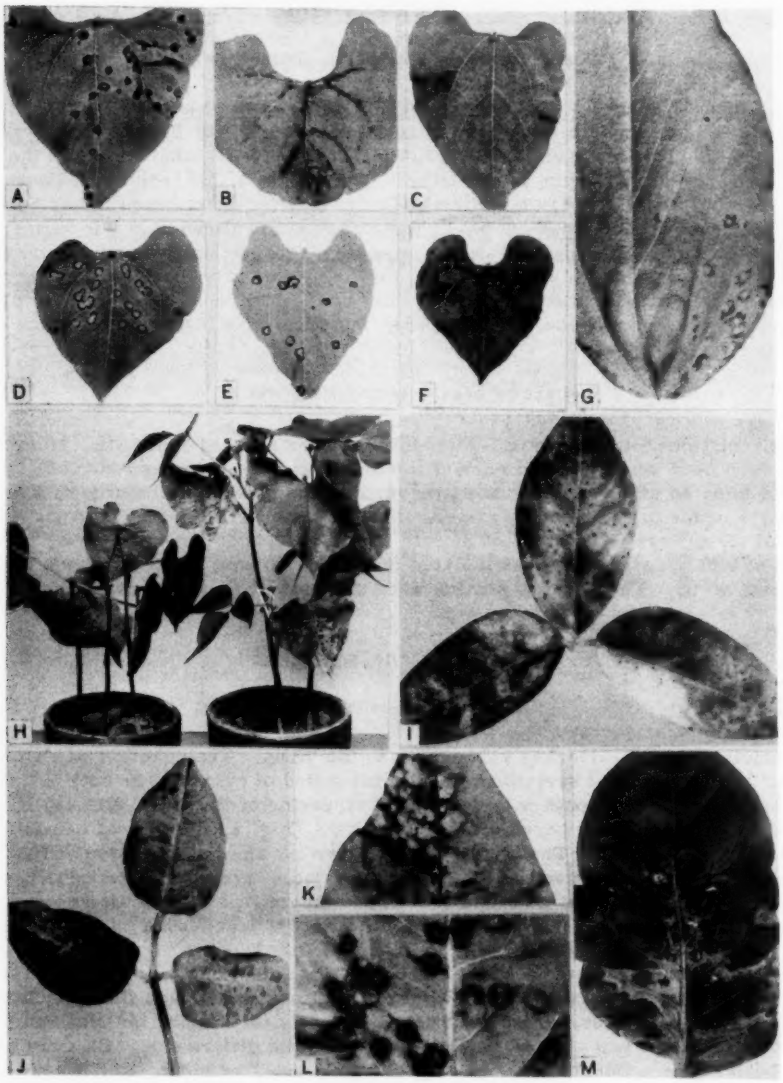

FIGURE 1. Symptoms produced by the bean strain and the McKinney strain of tobacco ringspot virus on various hosts. A-F, Local symptoms produced by the bean strain on bean varieties: A, Great Northern U. I. No. 16 (concentric rings), B, Lazy Wife (streaking of veins); C-F, Idaho Refugee (faint chlorotic spots), Blue Lake, Robust, and Pinto (streaking of veins and small necrotic spots), respectively. G, Systemic rings produced by the bean strain on Vicia faba. H, Symptoms produced by bean strain (left) and McKinney strain (right) on Low Champion. Note terminal killing and epinasty of primary leaves caused by the former virus. I, J, Systemic symptoms produced by McKinney strain and bean strain, respectively, on Phaseolus lathyroides. K, L, Local symptoms on Victor cowpea produced by bean strain and McKinney strain, respectively. M, Systemic symptoms on tobacco caused by the bean strain.

Table 1. Symptoms and reactions produced on bean varieties inoculated with four strains of the tobacco ringspot virus.

Table 1. Symptoms and reactions produced on bean varieties inoculated with four strains of the tobacco ringspot virus.

Variety	Bean strain		McKinney strain		Yellow strain		Green strain	
	Local symptoms[a]	Intensity of systemic symptoms	Local symptoms	Intensity of systemic symptoms	Local symptoms	Intensity of systemic symptoms	Local symptoms	Intensity of systemic symptoms
Black Creaseback	C, L, V, E	T	L, V	M	---	---	---	---
Black Valentine	C, L, V, E	S	L, V	N	L, V, E	S	C, L, E	T
Blue Lake	C, L, E	S	L	N	L	T	C, L	T
Bountiful	V, E	S	L	N	---	---	---	---
Brittle Wax	C, E	T	L, V, E	S	C, L, V, E	S	C, L, E	T
Flight	C, L, V	N	L, V, E	N	L, V, E	T	C, L	N
Florida Belle	C, L, V, E	T	L, V	T	---	---	---	---
Great Northern U.I.								
No. 16	L, V, E	S	L	N	L	N	L, V	T
No. 59	L, V	T	L, V	N	---	---	---	---
No. 81	L, V	T	L	N	---	---	---	---
No. 123	L, V, E	T	L, V	N	---	---	---	---
Idaho Pinto	L, V	S	L, V	M	L	M	C, L, V	N
Idaho Refugee	C, E	N	L, V, E	S	C, L, V, E	S	C, E	N
Lazy Wife	V, E	T	L	M	L, V	T	L, V	N
Low Champion	V, E	T	L	M	---	---	---	---
Michelite	L, V, K	N	L	M	---	---	---	---
Michigan Dark Red Kidney	C, L, E	N	L, V, E	S	C, L, V, E	S	C	N
Plentiful	C, L, E	T	L, V	T	---	---	---	---
Potomac	L, V, E	T	L, V	M	---	---	---	---
Red Kidney	C, L, V, E	T	L, V	T	---	---	---	---
Rival	C, L, E	N	L, V, E	S	L, V, E, K	S	C, L, V, E	T
Robust	L, K	N	L	M	L	M	L	M
Sensation Green Pod	L, E	T	L, V	N	---	---	---	---
Streamliner	C, L, V, E	T	L, V	T	---	---	---	---
Stringless Green Refugee	C, L, V, E	S	L	M	L	N	C, L, V	T
Topcrop	V	S	L	N	L, V, E	S	C, L, V, E	T
Unrivalled Wax	L	T	L	T	---	---	---	---

[a] C, chlorosis; E, epinasty of primary leaves; K, killing of inoculated leaves; L, local necrotic spots; V, vein streaking; S, very severe, death of infected plant; T, severe, with terminal killing; N, mild, with trifoliate necrotic spots; M, very mild, no systemic symptom.

Table 2. Symptoms produced by four strains of tobacco ringspot virus on selected hosts.

FAMILY Host	Bean strain	McKinney strain	Yellow strain	Green strain
LEGUMINOSAE:				
Canavalia ensiformis	Numerous necrotic local spots and streaking of veins; terminal killing and death.	Numerous necrotic local spots and streaking of veins; no systemic infection.	Same as the bean strain.	Numerous necrotic local spots and streaking of veins; systemic necrotic spots on trifoliate leaves.
Dolichos lablab	Scattered small necrotic local spots and some streaking of veins; late development of systemic necrotic spots on trifoliate leaves.	Similar to bean strain except for more numerous necrotic local spots.	Scattered small necrotic local spots and some streaking of veins; immediate development of systemic necrotic spots on trifoliate leaves and terminal killing.	Same as the bean strain.
Glycine max var. Lincoln	Necrotic local spots and streaking of veins; systemic mottling.	Necrotic local spots and streaking of veins; terminal killing and blackening of terminal node and stem; general mottling (1).	Similar to McKinney strain except for no blackening of nodes and stem.	Same as the bean strain.
Lupinus albus	Brownish necrotic local spots and concentric rings; systemic necrotic spots, terminal killing, severe necrosis and death of plants.	Large depressed chlorotic and necrotic local rings, yellowing of inoculated leaves; systemic necrotic spots, terminal killing and death of plants.	Brownish necrotic local spots and rings, yellowing; systemic necrotic spots, terminal killing and death of plants.	Chlorotic local spots with very small necrotic dots in aggregation, sometimes in rings; systemic mottling and chlorotic spots with necrotic dots at the center; no killing.
Phaseolus aureus var. Oklahoma 4347	Very few scattered red local spots, some forming pattern of a half-ring; systemic spots on trifoliate leaves.	Prominent large red necrotic local rings; terminal killing.		
Phaseolus lathyroides	Chlorotic local spots, vein clearing; systemic mottling.	Small necrotic local spots at center of chlorotic areas, vein clearing, marked yellowing and early drop of leaves; severe systemic necrotic spots, terminal killing and dwarfing of plant.	Same as the McKinney strain, but terminal killing more rapid.	Chlorotic local spots, vein clearing and yellowing of inoculated leaves; general mottling and recovery.

Table 2. (Continued) Symptoms produced by four strains of tobacco ringspot virus on selected hosts.

FAMILY Host	Bean strain	McKinney strain	Yellow strain	Green strain
Vigna sinensis var. Victor	Irregular reddening in a diffuse pattern, or forming a ring, streaking of veins; systemic necrosis and death.	Prominent large red concentric rings; terminal killing, stem necrosis and death.		
SOLANACEAE:				
Datura stramonium	Necrotic local rings and spots surrounded by chlorosis; systemic mottling, general chlorosis, and necrotic spots; early drop of young fruits.	Necrotic local spots; late and mild systemic development, few scattered rings, with normal fruit development.		
Nicotiana sylvestris	Faint local spots and whitish irregular lines; systemic faint rings and spots; recovery.	Small regular local concentric rings, more intense than bean strain; systemic irregular spots and zigzag patterns with necrotic borders; recovery.		
Petunia hybrida	Faint transparent local spots to whitish rings; systemic vein clearing on terminal young leaves; recovery.	Necrotic local frog-eye rings and rapid killing of inoculated leaves; severe systemic mottling, necrosis and leaf killing, slow recovery.	Large necrotic local concentric rings, yellowing and killing of inoculated leaves; severe systemic mottling and necrosis; slow recovery.	Chlorotic local spots and yellowing; systemic necrosis, much milder; recovery.
CUCURBITACEAE:				
Cucumis sativus	Chlorotic local spots on cotyledons; systemic faint yellow spots, general mottling.	Necrosis of inoculated cotyledons; systemic chlorotic spots, becoming necrotic, numerous necrotic spots on young leaves, leaf rolling and terminal killing.		
CHENOPODIACEAE:				
Spinacia oleracea var. Old Dominion	Slight streaking of veins and very mild chlorosis.	Depressed water-soaked local spots, later becoming necrotic and killing of leaves; systemic mottling, necrosis and killing.		

tested. In tobacco; cucumber, Cucumis sativus L.; muskmelon, C. melo L.; soybean, Glycine max (L.) Merrill; Phaseolus lathyroides L. (Fig. 1, I and J); petunia, Petunia hybrida Vilm.; and spinach, Spinacia oleracea L., the McKinney strain usually caused severe necrosis, while the bean strain was much milder in effect. In the Jack bean, Canavalia ensiformis (L.) DC., and jimson weed, Datura stramonium L., the reverse was true.

Other susceptible hosts showing less significant differences were moth bean, Phaseolus aconitifolius Jacq.; adzuki bean, P. angularis (Willd.) W. F. Wight; tepary bean, P. acutifolius Gray var. latifolius Freeman; scarlet runner bean, P. coccineus L.; urd bean, P. mungo L.; pea, Pisum sativum L.; crimson clover, Trifolium incarnatum L.; broad bean, Vicia faba L. (Fig. 1, G); vetch, V. sativa L.; Nicotiana glutinosa L., N. longiflora Cav., N. plumbaginifolia Viv.;zinnia, Zinnia elegans Jacq.; yellow crookneck squash, Cucurbita pepo L.; New Zealand spinach, Tetragonia expansa Murr.; beet, Beta vulgaris L.; China aster, Callistephus chinensis (L.) Nees; and guar, Cyamopsis tetragonolobus (L.) Taub. No infection was obtained on the following plants: white sweet clover, Melilotus alba Desr., pepper, Capsicum frutescens L., corn, Zea mays L., and sweetpotato, Ipomoea batatas (L.) Lam. Although red clover, Trifolium pratense L., showed no symptoms when inoculated with the bean and the McKinney strain, respectively, the bean virus was recovered from inoculated plants but the McKinney virus was not recovered.

Henderson and Wingard (2) demonstrated tobacco ringspot virus infection in potato, Solanum tuberosum L., and noted a marked difference in varietal susceptibility. In the present study the varieties Green Mountain, Irish Cobbler, Triumph, Spaulding Rose, and Pontiac were inoculated separately with the bean strain and the McKinney strain of the tobacco ringspot virus. After about ten days a few necrotic concentric ringspots appeared on inoculated leaves of Triumph variety inoculated with the bean strain. These spots gradually increased in size, finally reaching half an inch in diameter, and at the same time the surrounding tissue became yellow. None of the other varieties showed symptoms although the bean strain was recovered from Pontiac and Green Mountain varieties. It was not recovered from Irish Cobbler and Spaulding Rose. None of the varieties was infected with the McKinney strain.

Because of distinct differences in reactions to the different strains of the tobacco ringspot virus, several hosts are useful in differentiating these strains (Table 2). Phaseolus lathyroides (Fig. 1, I and J) is useful in that it manifests a very mild mottling when infected with the bean strain, but a severe necrotic reaction and terminal killing with the McKinney strain. Other species such as Canavalia ensiformis, Petunia hybrida, Glycine max, Nicotiana sylvestris, and Vigna sinensis var. Victor (Fig. 1, K and L) are also good differential hosts (Table 2). To differentiate the yellow strain from the others Dolichos lablab is useful, and to differentiate the green strain Lupinus albus (Table 2). Several bean varieties such as Idaho Refugee, Michigan Dark Red Kidney, and Stringless Green Refugee can also be used (Table 1).

Properties of the Bean Virus Strain

For the thermal inactivation and dilution tests, ten cowpea plants were inoculated for each treatment. The results were based on local lesion production. The thermal inactivation point for the bean strain was found to be between 65° and 70° C., and the dilution end point about 1 to 10,000. These data are similar to those reported for tobacco ringspot virus (2, 4, 5).

Repeated aging tests have shown that the bean strain loses its infectivity after 15 days at 18° C. This is the greatest longevity reported for any of the strains of the tobacco ringspot virus. Pierce (4) reported that the tobacco ringspot virus was infectious up to nine days, while McKinney (3), Pound (5) and Smith (6) reported that infectivity was lost after three to four days' storage.

Cross-Protection Test

Tobacco plants which had recovered from infection by the bean strain and previously non-inoculated healthy plants of the same age were inoculated with the McKinney strain of the virus. Four days later, abundant necrotic spots were produced on the "healthy" tobacco plants but none on plants previously infected with the bean strain. This indicated a strain relationship between the virus isolated from bean and the McKinney strain of tobacco ringspot virus.

SUMMARY

A new strain of tobacco ringspot virus was isolated from beans grown in Virginia and Delaware. It infects all bean varieties tested and produces on most of them more severe necrotic

symptoms than the McKinney strain of tobacco ringspot virus. However, on most plants tested other than bean the symptoms are milder than those produced by the McKinney strain.

Several hosts are useful in differentiating the several strains of tobacco ringspot virus: namely, Jack bean, hyacinth bean, soybean, white lupine, Nicotiana sylvestris, petunia, Phaseolus lathyroides, cowpea var. Victor, and three bean varieties (Idaho Refugee, Michigan Dark Red Kidney, and Stringless Green Refugee).

Cross-protection tests indicated a strain relationship between the bean strain and the McKinney strain of tobacco ringspot virus. The bean strain was inactivated between 65° and 70° C. and at about 1 to 10,000 dilution. It lost its infectivity at 18° C. after 15 days.

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EFFECT OF HEAT TREATMENT ON THE TYPE 2 VIRUS PRESENT IN THE ROBINSON STRAWBERRY[1]

R. H. Fulton

In attempting to find virus-free clones of strawberry varieties in Michigan, 238 strawberry plants from different sources were indexed to Fragaria vesca by the stolon graft method. From this extensive screening program virus-free sources were obtained for only three varieties. Type two[2] was the only virus found in these trials.

The stolon graft indexing procedure involves much time and space. A shorter approach to the problem of obtaining strawberry plants free from the type 2 virus prevalent in Michigan plantings would indeed be desirable. This prompted an investigation to determine whether the virus could be eliminated from the plants by heat treatment.

Materials and Methods

In February 1952, dormant Robinson strawberry plants infected with type 2 virus were brought into the greenhouse for treatment. The plants used in these tests were selected for uniformity of crown diameter and root systems. The plants were dipped for various periods in a water bath that was maintained within 0.5° C.± of a given temperature.

The exposure periods for all treatments were 5, 10, 20, and 30 minutes. The temperatures selected were 35°, 45°, 55°, and 65° C. Immediately after each treatment the plants were potted, and received daily care thereafter. Within a week's time, all plants treated at 55° and 65° temperatures were dead. The surviving plants developed stolons which were grafted to healthy indicator plants to ascertain the effect of the individual treatments on the virus. The indexing data obtained in this test are presented in Table 1.

Table 1. The effect of various exposure times at 35° and 45° C. on the type 2 virus in the Robinson strawberry.

Exposure time minutes	35° C. Number of plants	35° C. Effect on virus	45° C. Number of plants	45° C. Effect on virus
5	3	0[a]	3	0
10	3	0	3	0
20	3	0	3	0
30	3	0	3	0

[a] 0 indicates that plants when indexed to F. vesca transmitted the virus.

Results of this experiment, as given in Table 1, indicate that the virus was heat-stable in all strawberry plants treated at 35° and 45° C. for varying time intervals.

From the foregoing test it was apparent that the maximum sublethal temperature for the host plant would be between 45° and 55° C. It was then necessary to determine whether the inactivation point for the virus would come within this range.

The treatments were continued in temperatures ranging from 44° to 56° at 2° C. intervals. In the course of 14 days, it was evident that the heat treatments above 48° at 10 minutes exposure period were lethal to the strawberry plants. The virus inactivation data acquired on the remaining treatments are given in Table 2.

The data from Table 2 demonstrated that virus type 2 was not inactivated by the maximum temperature range for the host plant.

It is obvious from the data presented in this report that the strawberry type 2 virus present in the Robinson variety was not destroyed by heat treatments that were sublethal for the plants tested.

[1]Contribution 52-17, Department of Botany and Plant Pathology, Michigan State College.

[2]Demaree, J. B., and C. P. Marcus, Jr. 1951. Virus diseases of strawberries in the United States, with special reference to distribution, indexing and insect vectors in the East. Plant Dis. Reptr. 35: 527-537.

Table 2. Further studies on heat treatment of the Robinson strawberry carrying the type 2 virus.

Exposure time minutes	44° C. Number of plants	44° C. Effect on virus	46° C. Number of plants	46° C. Effect on virus	48° C.[b] Number of plants	48° C.[b] Effect on virus
5	3	0[a]	3	0	3	0
10	3	0	3	0	3	0
20	3	0	3	0		
30	3	0	3	0		

[a] 0 indicates that plants when indexed to F. vesca transmitted the virus.
[b] Maximum sublethal point.

Further investigations on other strawberry varieties and virus types may reveal thermal inactivation points. Meanwhile, the present method of stolon-grafting to indicator plants appears the most feasible for obtaining virus-free clones of strawberries.

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OCCURRENCE OF WESTERN ASTER YELLOWS VIRUS INFECTION IN GLADIOLUS IN EASTERN UNITED STATES

R. O. Magie, Floyd F. Smith, and Philip Brierley[1]

Gladiolus corms received by Florida growers from southern Oregon occasionally produce a few plants with spindly, weak, yellow leaves and short thin roots, a condition referred to by the growers as "grassy-top" and "hairy-root". This disease was first recognized in 1946 but remained too rare to attract much attention until January 1952, when a grower near Palmetto and another in Fort Myers, Florida, reported severe infection in several acres of the Picardy variety. The corms had been grown during the previous season in southern Oregon. When first examined, ten days after the corms had been planted, emerged shoots showed the typical grassy-top and hairy-root condition. After all plants had emerged, about two-thirds of them were found to be affected.

A few of the diseased plants were transplanted to a greenhouse at Bradenton, Florida, where they were grown in hydroponic culture to provide optimum growing conditions. The symptoms did not change under these conditions.

A third grower near Bradenton planted Hopman's Glory corms from the same Oregon grower and about 3 percent failed to emerge. Those that failed to emerge showed symptoms of the same type as Picardy except that the growth was much weaker. An Alabama grower reported that two-thirds of his plants from Picardy No. 2 corms were affected but very few from No. 1 corms, suggesting that corm size is reduced by first-season infection. These Picardy corms came from the same Oregon source, and the No. 1 and the smaller No. 2 corms were harvested from the same planting.

Discoloration or degeneration of vascular tissue was found near the base of all affected corms. In cross section, the core was larger and more irregular in shape than that of healthy corms of similar size (Fig. 1). The storage tissue at the basal part of corms was pale in color, coarse in texture, and bitter-tasting in comparison with healthy tissue. The base of the core was rough, hard, and black in many cases. Root traces were dark-colored and prominent and extended rather deeply into the corm. A few weeks after planting, small dark-colored warts were found on some corms which had developed small additional buds along the nodes.

Shoots from diseased corms of Picardy lived only two or three weeks in the field and were continuously replaced with weaker shoots. After two months few shoots grew more than 3 inches long. Root growth became weaker and sparse, but corms remained alive until harvest, five months after planting. Plants which were normal in January appeared healthy at harvest.

In January 1952, nine corms of Oregon-grown Picardy from a lot that had developed the disease in Florida field plantings were sent to the Plant Industry Station, Beltsville, Maryland, for diagnosis. Six corms produced normal plants, one was decayed by Fusarium without sprouting, and two expressed the typical grassy-top symptom. One of the last survived until September. From this plant the six-spotted leafhopper, Macrosteles fascifrons (Stål) (=M. divisus (Uhl.)), transmitted aster yellows to 5 of 10 China asters and to 4 of 5 healthy Picardy gladiolus test plants. Aster yellows virus was further transmitted from the infected China asters to 1 of 6 zinnias and to 4 of 5 celery plants. Zinnias displayed vein-clearing, rosetting, proliferation of lateral shoots and green flowers; celery showed yellowing, dwarfing, and stiff, upright, twisted leaves. Infection of zinnia and celery indicates the presence of the western strain of aster yellows virus, since these plants are known to be susceptible to the western strain but immune from the strain found in much of eastern United States.

Symptoms developed in the experimentally infected Picardy gladiolus are of special interest. Inoculated April 7 to May 9, when 8 to 10 inches tall, by May 16 these plants showed yellowing, bleaching, and drying of the youngest leaf at the site of leafhopper feeding, immediately above the juncture with the next older leaf. These inoculated leaves subsequently turned completely yellow and withered. The plants made no growth after inoculation. By June 20 the older leaves had turned dull yellow, closely stimulating the leaf symptoms of Fusarium yellows. On July 8 the four affected plants were dead, the old corms were soft and depleted, and new corms and contractile roots were lacking. The one plant that had escaped infection was then in flower, and had developed many contractile roots and a new corm larger than the old one. Thus aster yellows virus induces a lethal disease in gladiolus when plants become infected in the early stages of growth. In-

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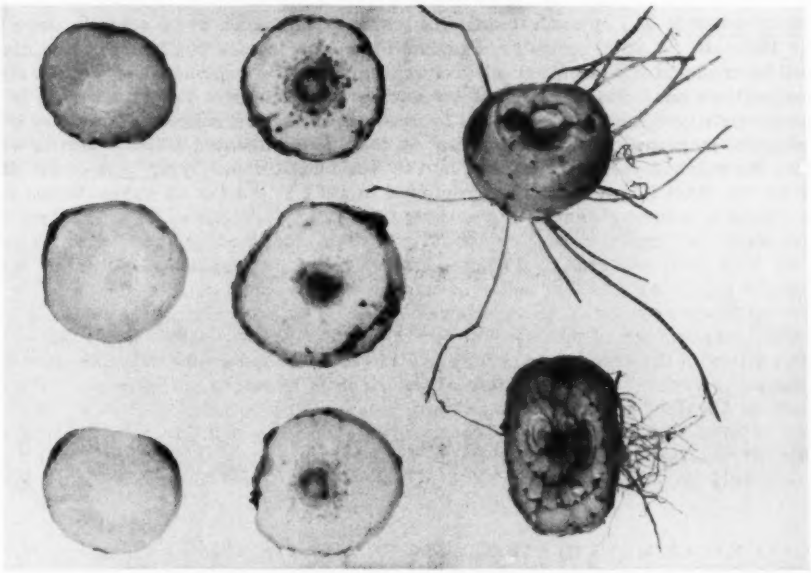

FIGURE 1. Second-season symptoms of western aster yellows in corms of Picardy gladiolus. At left, cross-sections of 3 healthy corms with normal yellow color and water-soaked tissue. In center, cross-sections of affected corms showing colorless storage tissue, darkened vascular tissue, and enlarged cores. At upper right, healthy corm with shoot removed, showing normal roots. At lower right, affected corm with hairy root and a very large number of buds (shoots removed).
Photograph by R. O. Magie.

fected plants that survive to the following season and then express the symptoms known as grassy-top presumably must be inoculated in later stages of growth.

In April 1952 Mr. A. H. Draughon of Fort Myers, Florida, supplied additional corms of Oregon-grown Picardy which he considered to be affected with grassy-top. Of 182 corms planted in the greenhouse at Beltsville on April 21, only one produced a normal plant. All others expressed grassy-top. Mr Draughon thus had made a nearly perfect separation of diseased corms from a mixture with normal ones on the basis of the warty appearance associated with development of additional lateral buds. On July 9 the grassy-top plants had many sprouts with thin yellowish leaves 6 to 12 inches long. The one normal plant developed a single shoot 36 inches tall but failed to flower.

On April 18 the Oregon grower supplied us 23 Picardy corms of unselected stock similar to that sold to the Florida growers. Thirteen of these produced normal single shoots and flowered July 15 at a height of 36 to 40 inches. The remaining 10 plants developed three to seven shoots per corm with thin yellowish leaves 6 to 10 inches long. The disease tends to be self-eliminating, but many of the affected plants are able to persist weakly for four to eight months or possibly longer under glass.

There was no evidence of spread of the disease in Florida. Wild hosts of aster yellows virus were examined in the vicinity of the Picardy planting near Palmetto by the writers and by W. N. Stoner of the Everglades Experiment Station, Belle Glade, Florida, late in April. Stoner examined weeds and made leafhopper collections again in July. Definite symptoms of aster yellows infection were not seen. Samples of weeds collected at Piney Point, Florida, by Magie and Stoner were mailed to Beltsville and tested for presence of aster yellows virus by leafhopper transfer to China aster. None of these weeds had symptoms characteristic of aster yellows, but some did show yellowing of upper leaves. No virus was detected in any of these plants including ragweed (Ambrosia artemisiifolia L.)[2], wormseed (Chenopodium ambrosioides L.), and fleabane

[2]The writers are indebted to S. F. Blake and Erdman West for plant determinations.

(Erigeron canadensis L.) by such tests. No leafhoppers known to be vectors were found in the area near Palmetto in April or July. Further examinations for leafhoppers and also for infected plants will be made in cool weather when symptoms may be expressed more clearly.

Aster yellows has been recognized previously in gladiolus. The type strain in the East has been experimentally induced in gladiolus by leafhopper transmission[3]. Here too the experimentally produced symptom was a "grassy-top" in the second season, but the leaves were green in contrast to the marked yellowing caused by the western strain. Symptoms of the disease in gladiolus on the West Coast were first reported in 1952[4]. As far as known to the writers, the losses in Florida in the present year represent the first outbreak of aster yellows as a gladiolus disease of economic importance. Previously disease incidence had been seen to range from a trace to the 2 percent reported by Palm and Young [4]. As a disease of gladiolus, aster yellows may assume a minor role except under special weather conditions, such as severe drought that forces the leafhopper vector to leave other food plants and to congregate on irrigated gladiolus.

The chief importance of the introduction of western aster yellows in gladiolus into eastern United States lies in the menace to celery and zinnia in areas where only the eastern strain of the virus is known to occur. Neither strain of the virus is known to occur in southern Florida; so introduction of diseased plant material should be avoided if at all possible. Celery has been free from aster yellows in Eastern States because this crop is immune from the type strain of aster yellows already established. Zinnia, which escapes yellows in Eastern States for the same reason, is widely grown by amateurs who have abandoned China aster.

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A GLASS AND PLASTIC INSECT-PROOF CAGE[1]

Hurley Fellows and Richard V. Connin[2]

Experimental work with a specific insect often is made more difficult by contamination with other insects or arachnids. Obviously, it is essential that no contamination occur when attempting to transmit specific virus diseases with insects. Many cages in use permit the passage of undesired insects. The essentials of any insect-proof cage for both plants and insects are that it

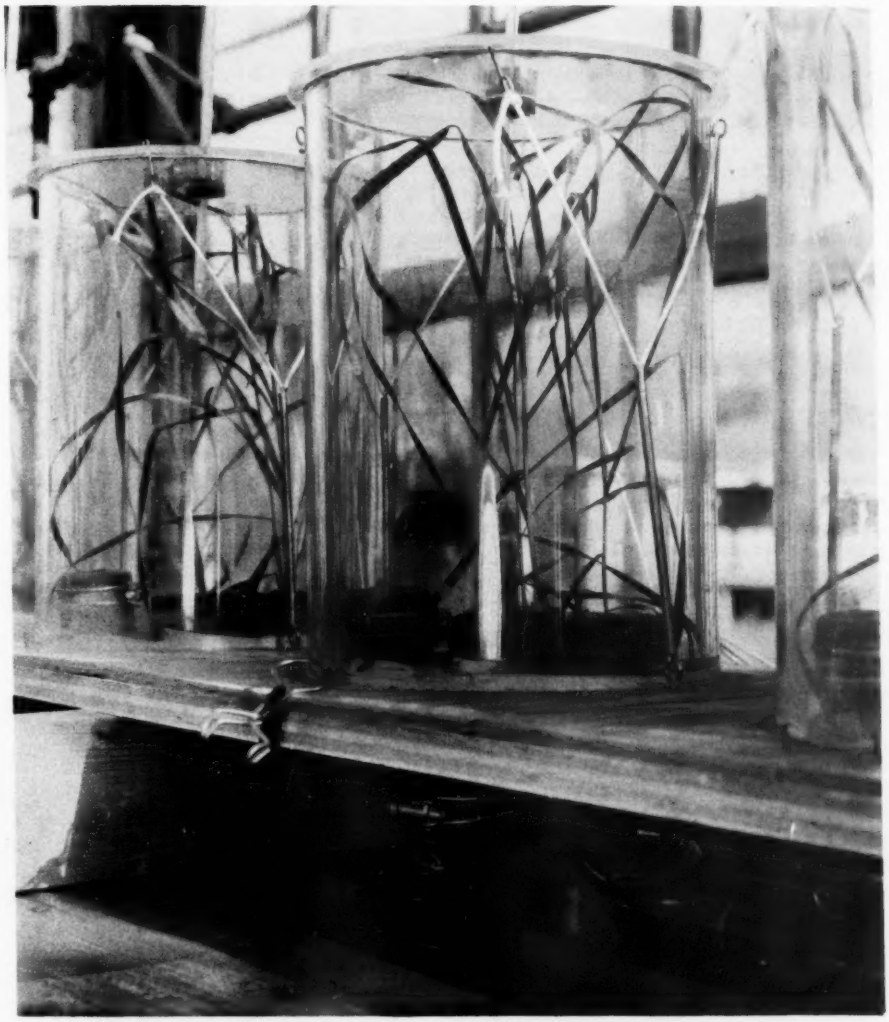

FIGURE 1. Enlarged view of insect cages used in virus transmission studies. This shows the plants and pots in place, the watering apparatus and the air intake.

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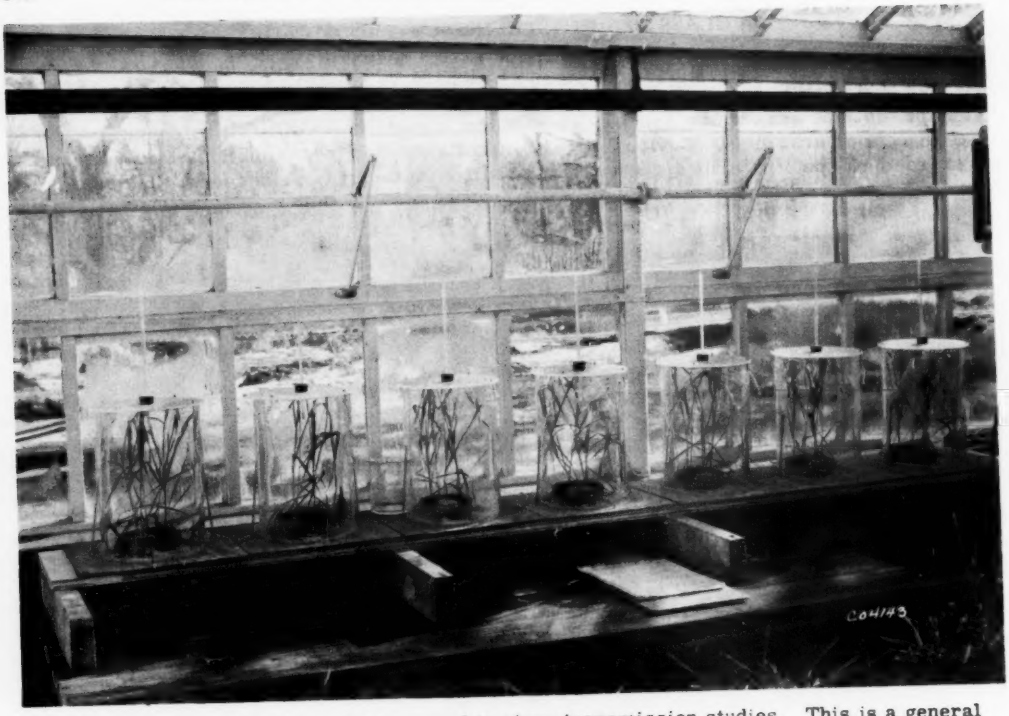

FIGURE 2. Insect proof cages used in virus transmission studies. This is a general view showing a complete battery of cages.

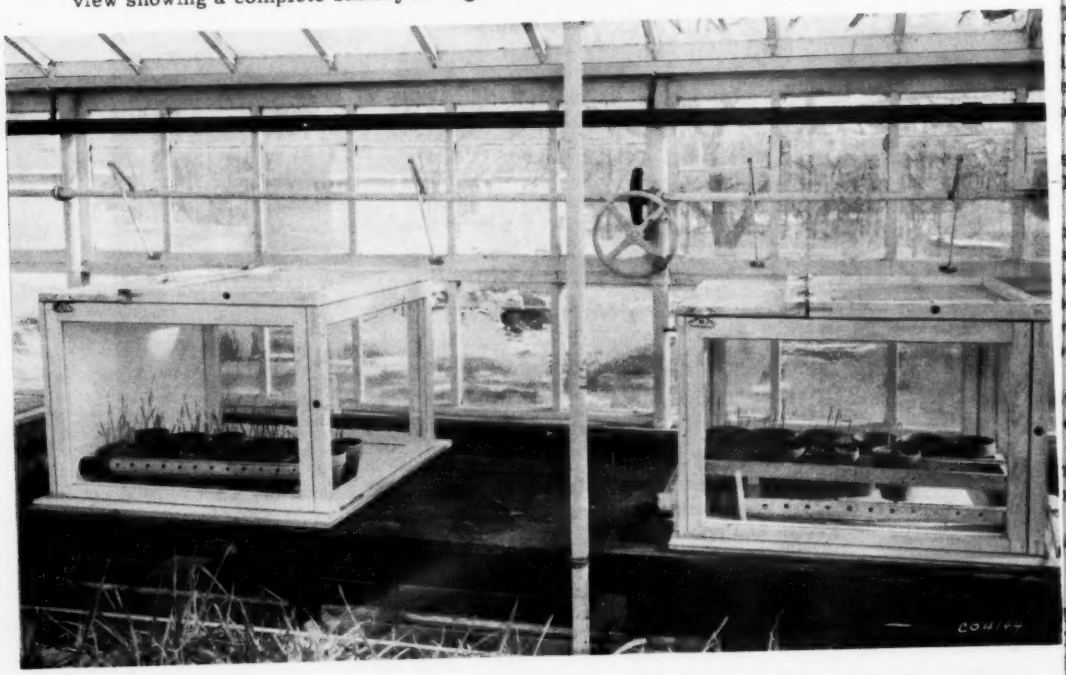

FIGURE 3. Auxiliary rearing and observation chambers used in virus transmission studies.

be adequately lighted and ventilated and that it maintain a favorable temperature. The cage described in this paper fulfills these requirements. It was built for use in attempting to find the vector of yellow streak-mosaic of wheat. One season's work has proved the practicability of this type of cage. Plants grown in the cages have been normal in appearance and introduced insects have thrived. Thus far no contamination has occurred.

Description of Cages

The individual cage consists of a ground edged Pyrex glass tube 8 inches in diameter and 12 inches long. These tubes are placed seven in a line on a common base of 5-ply plywood 12 inches wide, 1 inch thick and 8 feet long. Each is held in place by a collar of 3-ply plywood fastened to the base. The bottom of each tube is sealed into its collar with Armstrong's Caulking compound, which remains pliable but is not sticky after 24 hours of exposure. Three holes are bored in the base under each tube, two for ventilation and one for the pot in which plants are growing. Ordinary 1 1/2-inch by 3-inch pipe nipples are screwed into the ventilating holes extending below the baseboard. The hole for the pot is beveled to fit an ordinary 4-inch clay pot just below the top rim. When in place, the top of the pot is in the cage and the lower part protrudes through the baseboard. The pots are sealed in place with caulking compound. The top of each tube is closed with a Plexiglass disk (acrylic plastic) 3/16-inch thick. The disk, which is 9 inches in diameter, extends 1/2-inch beyond the rim of the cylinder. These disks are sealed to the cylinders with caulking compound and further secured from accidental disturbance by small spiral springs maintained under tension between the edge of the disk and the base. A 1-inch hole in the center of the disk is plugged with a perforated rubber stopper that holds a thermometer extending into the cage. The stopper may be removed to introduce any desired insect.

The pots are watered through a copper tube that extends up through the hole in the bottom of the pot to a point slightly above the soil surface. The hole in the pot is sealed with a short piece of rubber tubing around the copper tube where it passes through. Water poured into a funnel passes through about 2 feet of rubber tubing through the copper tube and up above the soil surface. The outer end of the rubber tube is closed with a pinch cock except during the watering.

Ventilation is provided by a Dayton blower with a capacity of 150 cubic feet of air per minute. The air passes to each cage through a sheet metal manifold measuring 4 x 4 inches and approximately the length of the baseboard. Tubular outlets soldered on the manifold are connected to the inlet nipples of each cage with flexible automobile radiator hose 1-3/4 inches in diameter. Dampers in each hose are used to regulate air velocity. The exhaust air passes through the outlet nipple into the greenhouse. (Figures 1 and 2.)

The air inlets and outlets and the blower inlets are double-screened with 36-mesh lumite screen. As an additional precaution, the interior of each outlet and inlet nipple is coated with Tanglefoot to catch crawling insects, spiders or mites.

Two large insect-proof auxiliary chambers also were built. These were made from single-pane window sash except that the bottom and one side are of 5-ply plywood. The joints are sealed with felt weather stripping and caulking compound. The parts are fastened together with lag screws. Ventilation is provided as in the small cages. Watering is done by a long movable piece of copper tubing inserted through a stoppered hole in the wood side of the cage. The exhaust vents are also in this side. A removable section in the top permits the passage of pots in and out. (Figure 3.)

One of these auxiliary chambers is used prior to insect transmission attempts. Plants are started in this chamber and later are placed in the small cages. They are grown in heat-sterilized soil in 4-inch pots which have the copper watering tubes already in place.

The other chamber is used for observation. After plants have been exposed to the feeding of insects in the small cages, they are placed in this chamber. The glass sides afford opportunity to observe any symptoms which the plants may show.

Between experiments both the chambers and cages are sterilized with a fumigant or by washing with 70% ethyl alcohol.

IVISION OF CEREAL CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND GRICULTURAL ENGINEERING; DIVISION OF CEREAL AND FORAGE INSECT INVESTIGATIONS, BUREAU OF ENTOMOLOGY AND PLANT QUARANTINE, UNITED STATES DEPARTENT OF AGRICULTURE; AND KANSAS AGRICULTURAL EXPERIMENT STATION, COOPERTING

CURVULARIA ON GLADIOLUS IN OREGON

C. G. Anderson, Roy A. Young, and E. T. Palm[1]

The Oregon Agricultural Experiment Station did not participate in the 1952 national cooperative gladiolus corm treatment trials sponsored by the American Gladiolus Council and the North American Commercial Gladiolus Growers because the corm lots to be distributed for the tests were reported to be infected with Curvularia sp. and the pathogen had not been found in Oregon.

In December 1951 a few corms infected with Curvularia were found in a lot of the variety Cotillion in one storage in southern Oregon and in a lot of Pellegrini at another. In February and March 1952, Curvularia-infected corms were found in a lot of Spic and Span and in an additional lot of Cotillion. Only a few infected corms were found in each instance and the disease has not been observed on gladiolus foliage or flowers.

The isolates of Curvularia obtained from gladiolus corms in Oregon fit very closely Boedijn's description of Curvularia lunata (Wakker) Boedijn (1), and the characteristics listed by Groves and Skolko (2) for an isolate from onion seed from California and an isolate from pepper seed from Michigan. Conidia were brown, 8-12 by 24-30μ, 3-septate with the third cell from the base much larger and darker than the other cells (Figure 1). Decayed areas on affected corms were shallow, dark brown with a rough, somewhat eroded appearance (Figure 2), and frequently were partially delimited by the leaf scars (upper right and lower center Figure 2).

FIGURE 1. Conidia and conidiophore of Curvularia isolated from corms of Cotillion gladiolus. X600
Photograph by L. E. Jones.

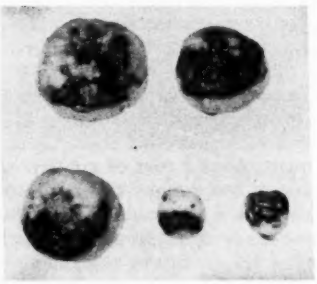

FIGURE 2. Curvularia lesions on Cotillion corms. X 3/8.
Photograph by H. H. Millsap.

Although Curvularia causes a serious foliage and flower disease on gladiolus in Florida (4), and in other locations in Eastern and Southern States (3), the disease is not yet of economic importance in Oregon. It is probable that the low humidity in most gladiolus production areas in Oregon will not favor extensive development of this disease.

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SOIL FUMIGATION WITH CHLOROBROMOPROPENE FOR CONTROL OF SCLEROTIUM ROLFSII IN DUTCH IRIS

Frank A. Haasis

Crown and stem rot, caused by Sclerotium rolfsii Sacc., is an important disease of Dutch iris in southeastern North Carolina owing to the fact that many sites suitable for iris culture are infested, and the humid and warm climate is ideal for fungus development.

The disease appears by mid-May and increases at an alarming rate through the summer months. Commercial growers have learned that early harvest of their bulbs reduces loss from this disease but at some sacrifice in the form of reduced yields and probably, also, in bulb quality which is subsequently reflected in less than maximum varietal performance.

This paper reports an experiment performed at the Vegetable Research Laboratory near Wilmington, North Carolina, to determine whether or not the chemical chlorobromopropene (Shell CBP)[1] would markedly reduce or eradicate this soil organism.

Methods

A well-drained field that had borne two successive crops, tomatoes and shallots, which were heavily infected with S. rolfsii, was selected for the experiment. The field was laid off in four blocks, each containing four plots 18 x 100 feet. The large plots made possible the use of tractor-mounted equipment for applying the chemical.

The chemical was injected 6 inches deep in rows 12 inches apart by means of an "Insco pressure applicator"[2] with six outlets fitted to chisel plows mounted on a tractor cultivator. Row applications were made with the same equipment by using two injectors spaced 3 feet apart.

The following treatments were applied to the soil after it had been tilled and disked until ideal for planting:

(1) Chlorobromopropene at rate of 15 gal./A. applied in rows 1 foot apart on October 19, 1950.

(2) Chlorobromopropene at rate of 15 gal./A. applied in rows 1 foot apart on October 19, 1950. The soil was spaded prior to a second application of chlorobromopropene at 15 gal./A. in rows 1 foot apart on October 31, 1950.

(3) Chlorobromopropene at 15 gal./A. applied in rows 1 foot apart on October 19, 1950. The soil was spaded prior to a second application of chlorobromopropene at 5 gal./A. applied in rows 3 feet apart on October 31, 1950.

(4) No soil treatment.

Calibration of the equipment, at intervals, revealed that rates of application were approximately 10 percent in excess of those originally planned. Furthermore, variation was encountered between injection outlets, but none fell below the originally planned gallonage per acre.

In order to prevent excessive escape of fumigant from the soil surface, each plot was compacted with a drag immediately after treatment. Three hours after the first treatment was made a heavy precipitation (2.5 inches) also provided a water seal. This amount of rain, however, presumedly interfered with dispersion of the gaseous phase of the chemical through the soil. The soil temperature, at time of treatment, was 65° F. at a depth of 6 inches.

Iris bulbs of Imperator and Wedgewood varieties were planted on November 10, 1950. Prior to planting, one half of the bulbs of each variety was steeped in a 1% formalin solution for 30 minutes. Hence, the final field lay-out consisted of four soil treatments split for varieties and again for bulb treatment, and each of these replicated four times. There were four test rows per plot thus allowing for a treated border area, 3 feet wide, surrounding every set of rows.

Fertilization and cultivation procedures were the same as those of commercial practice. All plots were harvested by hand to minimize loss of bulbs.

Results

Two criteria were used in evaluating treatment effects, namely: (1) the percentage of bulbs infected and infested with Sclerotium rolfsii at harvest, and (2) total bulbs harvested per plot.

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[1] Shell Chemical Company

[2] Insect Control Sales and Service, Charlotte, N. C.

Table 1. Soil fumigation with chlorobromopropene for control of Sclerotium rolfsii as influenced by variety and bulb steep in formalin at planting. Data expressed as mean percentage of bulbs with signs of S. rolfsii at bulb harvest.

Soil treatment			Varieties			
First application 10/19/50	Second application 10/31/50	Bulb treatment	Imperator	Wedgewood	Mean for soil X bulb treatment	Mean for soil treatment
15 gal./A.	15 gal./A.	Formalin	11.5	15.4	13.4	13.7
		None	11.3	16.7	14.0	
15 gal./A.	5 gal./A.	Formalin	5.6	13.3	9.4	8.1
		None	2.6	10.9	6.7	
15 gal./A.	None	Formalin	10.3	28.3	19.3	15.7
		None	11.5	13.0	12.2	
None	None	Formalin	37.2	37.4	37.3	32.0
		None	23.6	29.8	26.7	
					Non sig.	L.S.D. .05=13.7
					Mean effect for bulb treatment	
Mean effect for variety X bulb treatment		Formalin	16.1	23.6	19.9	
		None	12.2	17.6	14.9	
			non sig.		non sig.	
Mean effect for variety			14.2	20.6		
			L.S.D. .01=4.991			

Partial control of S. rolfsii was achieved with injections of chlorobromopropene as evidenced by comparing the main effects of the soil treatments, Table 1. Infection and infestation levels were significantly lower in each of the treated plots as compared with the untreated control. Although the three levels of chemical failed to show a significant departure one from the other, the trend toward least infection and infestation was in direct proportion to the amount of material applied or to its proximity to the soil environment of the iris plant. The data also indicate that Wedgewood was more affected by S. rolfsii than was Imperator. Treating the bulbs in a formalin steep, prior to planting, failed to control S. rolfsii. Likewise, all interaction effects were insignificant.

The results obtained by evaluating treatments from the standpoint of total bulbs harvested present an entirely different picture. Soil treatments did not affect the numbers of bulbs; hence indicating that under the conditions of this test a phytocidal level was not reached. Bulbs steeped in formalin, however, outyielded those not treated. A difference in varietal performance was evident, which may or may not be real, since there were fewer Imperator bulbs planted per plot than Wedgewood.

Of greater interest than any of these main effect differences or conformities is the highly significant interaction effect involving soil vs. bulb treatments, shown in Table 2. Bulbs steeped in formalin, prior to planting, appeared to yield as of one population whether or not they

Table 2. Effect of soil treatment with chlorobromopropene, bulb treatment in formalin, and variety on bulb harvest. Data expressed as mean number of bulbs harvested.

Soil treatment			Varieties			
First application 10/19/50	Second application 10/31/50	Bulb treatment	Imperator	Wedgewood	Mean for soil X bulb treatment	Mean for soil treatment
15 gal./A.	15 gal./A.	Formalin	180	799	489	394
		None	125	473	299	
15 gal./A.	5 gal./A.	Formalin	171	781	476	353
		None	108	351	229	
15 gal./A.	None	Formalin	163	702	432	422
		None	170	652	411	
None	None	Formalin	188	734	461	437
		None	147	679	413	
					L.S.D. .01= 101.79	non sig.
					Mean effect for bulb treatment	
Mean effect for variety X bulb treatment		Formalin	176	754	465	
		None	137	539	338	
			L.S.D. .01=89.17		L.S.D. .01= 50.90	
Mean effect for variety			156	646		
			L.S.D. .01=63.04			

were planted in soil receiving different levels of chlorobromopropene or even in soil receiving no treatment. Non-treated bulbs planted in non-treated soil or in soil treated with a single application of chlorobromopropene at the rate of 15 gal./A. (incomplete fumigation?) likewise survived in approximately the same proportion as did those from the formalin-treated bulbs. But, untreated bulbs planted in soil thoroughly fumigated with chlorobromopropene survived at a significantly lower level. This phenomenon is explained by the fact that most of this loss in stands was due to the basal rot disease caused by Fusarium sp. (probably F. oxysporum Schlecht.). Hence, it appears that the Fusarium organism was carried on the bulbs at planting; that a preplanting steep in formalin destroyed the fungus and excluded it from the soils; that natural Fusarium inhibitors were present in the untreated and incompletely fumigated soil which held the organism in check in the absence of formalin steep; but in the absence of these inhibitors (in the adequately fumigated soil) and with the introduction of Fusarium on the bulbs at planting, the organism was unimpeded and hence many of the bulbs were infected and failed to survive. If this interpretation of the facts is correct it would then appear that the Fusarium inhibitors are of biological origin, and furthermore that the inhibitory effect is maintained only in an environment with the organism or organisms producing the inhibitor.

As mentioned previously, an interpretation of the significant varietal difference for bulb survival is invalidated because of different planting rates. The significant interaction effect

(variety x bulb treatment), however, does appear to be real (Table 2), for the effect of formalin treatment in Wedgewood is more marked than it is with Imperator. As further evidence, bulb rot, due largely to Fusarium and accruing during the subsequent storage period, amounted to 52 percent for Imperator and 28 percent for Wedgewood with a required L.S.D. (.01) of 7.47. This would tend to indicate that Imperator was more susceptible to Fusarium than Wedgewood, but not unequivocally, since the previous history of the two stocks was not of a parallel nature. The significance, therefore, of the interactive effect involving iris variety and bulb treatment cannot be resolved with certainty with the data at hand.

Summary and Conclusions

The results of this study show that soil populations (based upon disease incidence) of Sclerotium rolfsii can be reduced by fumigation with chlorobromopropene. Also borne out in this study is evidence that thorough soil fumigation with chlorobromopropene in some instances may indeed be harmful when inadvertently a second pathogen is introduced.

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TWO TYPES OF ANTHRACNOSE RESISTANCE IN CUCUMBERS

W. C. Barnes and Wm. M. Epps

Anthracnose (Colletotrichum lagenarium (Pass.) Ell. & Halst.) was not considered a serious disease of cucumbers in South Carolina prior to introduction of the downy mildew-resistant variety, Palmetto (1). The disease was seldom reported on the spring crop and was very rarely observed to cause any appreciable damage because anthracnose did not attack the crop until late, and downy mildew (Pseudoperonospora cubensis (Berk. & Curt.) Rostow.), which usually appeared about the same time or somewhat earlier, killed the crop before it could become well established. Since downy mildew-resistant varieties such as Santee prolong the spring harvest season, greater damage from anthracnose may be expected. A few areas in the South have occasionally reported extensive losses in recent years.

As soon as Palmetto made possible extensive fall plantings in South Carolina and neighboring States, anthracnose became recognized as a potentially serious disease. The high temperatures and frequent rainy periods that prevail from planting time in August to harvest in October are very favorable for the pathogen. Volunteer watermelons and cucumbers provide abundant inoculum, and every year some fields are lost because of this disease. The disease appears to be universally present, and only by destroying volunteer plants and following a good control program with a fungicide such as zineb (2) can serious losses be avoided. Even then the disease sometimes becomes epiphytotic during hurricane periods when fungicides cannot be applied.

After the potentially serious nature of anthracnose was recognized, hundreds of cucumber varieties and foreign introductions were screened in a search for resistance. Susceptibility to anthracnose in commercial varieties varied slightly, but none showed more than a slight tolerance to the disease. For example, Marketer was extremely susceptible; while Palmetto showed a slight degree of tolerance.

Two types of resistance were found among the accessions received from the Division of Plant Exploration and Introduction. Six accessions of the hundreds tested in greenhouse and field tests showed a sufficient degree of resistance to be worth while in a breeding program. One of these, (P. I. 197,087), appeared to be immune to the anthracnose cultures used. Preliminary data, based on a limited number of plants, indicate that resistance is only partially dominant in the F_1 and backcross generations. The F_2 segregated in a ratio of approximately 1 resistant: 2 intermediate: 1 susceptible. The degree of resistance of the F_2 resistant plants varied considerably and very few, if any, plants showed resistance equal to that of the resistant parent. It therefore appears that resistance is controlled by more than one gene. Further crosses of P. I. 197,087 with several slicing and pickling lines have been made and additional data on the mode of inheritance will be available in the near future.

Five other accessions (P. I. Nos. 175,111; 175,120; 179,676; 183,308; and 183,445) showed a high degree of resistance although much less than that shown by 197,087. When seedlings were inoculated, in the field or greenhouse, susceptible plants were seriously damaged or were killed outright. Small lesions developed on the resistant plants of these five lines about two days after symptoms first appeared on the susceptible lines. These lesions enlarged very slowly and remained quite small and apparently inactive until senescence began, at which time they enlarged rapidly. Very little secondary spread occurred during the time the plants were growing actively, whereas spread on adjacent susceptible plants during the rainy periods was extensive.

The resistance of these five accessions appeared to be inherited as a single dominant factor, as has been reported with anthracnose resistance in the watermelon (Layton 1937) (3). These five anthracnose-resistant accessions were crossed and the progeny backcrossed with some of the South Carolina downy mildew-resistant lines. Rapid progress has been made in the production of good fruit types resistant to both diseases. Some of these should be ready for testing by 1954.

All six resistant accessions are extremely late, flowering two to four weeks later than commercial varieties. They produce vigorous vines and fair to poor type fruit, and all are at least moderately resistant to downy mildew. In preliminary tests the fruit of these resistant lines did not appear to have any resistance to anthracnose infection.

It thus appears that two distinct types of resistance to anthracnose in cucumbers have been located. The two types differ, not only in the degree of resistance exhibited, but also in the mode of their inheritance.

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CLADOSPORIUM ROT OF PEPPERS

G. B. Ramsey and Barbara C. Heiberg

For many years a decay of California bell type peppers has caused serious damage to some shipments received on the Chicago market during fall and early winter. The disease is caused by Cladosporium herbarum Fr. Moderately cool, wet seasons when rain or fog keeps the plants and fruits moist seem to favor infection and development of this decay. Shipments received after six days in transit usually show only slight evidence of infection but decayed shipments or lots of peppers held on track or in stores develop conspicuous spots within a few days (Fig. 1 A). Infected peppers held in store coolers (36° to 40° F.) for a week or more develop serious rot (Fig. 1 B). On arrival or soon thereafter the infected areas appear as light tan spots 1.5 to 3 mm. in diameter. As the spots enlarge they become slightly sunken and have a dark center and a light-brown border. With further development the central area becomes black and shiny and the brown border sharply delimited. The decayed tissue under spots 5 mm. in diameter and larger is light grayish-brown and rather spongy in texture, making it easily removed with the point of a knife or a pencil. In a very humid atmosphere a fine green mold made up almost entirely of spores develops over the larger lesions. Cultures made from spots of various size produced colonies typical of C. herbarum.

FIGURE 1. Rot of pepper caused by Cladosporium herbarum. A, conspicuous spots such as develop after a few days in a store; B, serious rot on infected pepper held a week or more in a store cooler (36-40° F.)

In 1932 a similar decay of California tomatoes was described and illustrated by Ramsey and Link (2), but at that time it was thought that the causal organism was a strain of C. fulvum Cke. Further studies and comparison of a large number of isolates of Cladosporium from peppers and tomatoes show that the decay is induced by a strain of C. herbarum. The only other reference to this species on tomatoes known to the authors are those of Perotti and Cristofoletti (1), who reported a fruit rot of tomatoes in Italy with which it and a new species of bacteria were associated, and a recent mention of its occurrence on tomatoes in Norway by Ramsfjell (4). A recent publication by Ramsey, Wiant, and McColloch (3) appears to be the only one that mentions parasitism of C. herbarum on peppers.

Cladosporium rot has been found following injuries in peppers and also associated with other diseases such as Botrytis rot and Alternaria rot, but in most instances lesions appear where there are no visible wounds and no other organisms are present. At first it was thought that germinating spores contaminating the fruits penetrated stomata but a careful examination revealed that there are no stomata in the epidermis of pepper fruits. The calyx and stem have an abundance of large stomata spaced at intervals of about 200 microns and infections have been observed in these parts. However, infections of the fruit are more common.

In laboratory experiments inoculations through wounds and by spraying a water suspension of spores on apparently uninjured peppers caused infection. In a typical experiment 12 peppers

that were inoculated with spores and mycelium in slight wounds and held at 40° F. developed lesions averaging 3.8 mm. in diameter in three weeks and 10.2 mm. in six weeks. Inoculations by means of a spore suspension sprayed on apparently uninjured peppers produce typical lesions but at a slower rate than on wounded fruits. The rate of development of Cladosporium lesions resulting from natural infection is shown by the following measurements. In one commercial lot of peppers 310 spots that averaged 2.3 mm. in diameter on arrival averaged 3.8 mm. after four days in storage at 40°. Another lot of peppers with 26 spots averaging 1.2 mm. in diameter on arrival averaged 2.7 mm. after three days in storage at 70°, and during this time several new light tan spots about 1 mm. in diameter developed.

C. herbarum grows slowly on potato dextrose agar plates. At 35° F. no visible growth occurs for about nine days and thereafter growth is very slight. Colonies average 6.7 mm. in diameter at 40° in seven days; 16.5 mm. at 50°; 27.6 mm. at 60°; and 31.5 mm. at 70°. The minimum temperature for growth on this medium is between 32° and 35°, the optimum 70°, and the maximum about 85°.

Since C. herbarum is a weak parasite of peppers and seems to cause infections only under special conditions of temperature and moisture it appears unlikely that serious development of decay induced by this organism will occur commonly in shipments of this commodity. However, under field conditions favorable for contamination and infection of the fruit, serious loss may occur in shipments before the peppers can be marketed. In October 1952 a shipment of 245 crates of California peppers was accepted on delivery as satisfactory by a Chicago receiver and 50 crates were sold. The 195 crates remaining were placed in a cooler at 40°. Five days' later an inspection showed 90 to 100 percent of the peppers in representative crates to be severely spotted with Cladosporium lesions. Some peppers had over 50 spots, ranging from 1 to 8 mm. in diameter.

Although refrigeration of shipments of peppers below 40° will retard the development of Cladosporium rot, it will not completely check the decay. The development of field or packinghouse treatments together with the careful grading out of all spotted fruits at loading time appears to be the best method of controlling this disease.

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REACTION OF NICOTIANA SPP. TO THE LONG ISLAND, NEW YORK GOLDEN NEMATODE OF POTATOES, HETERODERA ROSTOCHIENSIS

Julius Feldmesser and George Fassuliotis

Various workers in the past 23 years have investigated the possibility that Heterodera rostochiensis completes its life cycle on plants other than potato and tomato. Reinmuth (5), testing various members of the Solanaceae, found potato and tomato to be the only susceptible plants. Triffitt (7) reported Nicotiana, Atropa, Petunia, Salpiglossus, Physalis, Browallia, Solanum crispum, and S. capsicastrum to be negative. Goffart (1) tested Nicotiana sylvestris and found no evidence of susceptibility. Negative results were obtained for eggplant, pepper, and tobacco by Mai and Lownsbery (3). Jones (2) reported negative results with four species of Nicotiana, namely, N. tabacum, N. sylvestris, N. paniculata, and N. langsdorffii. Oostenbrink (4) also conducted host-range studies. He reported negative results for Nicotiana, Datura, and Atropa but he considered these results inconclusive.

In September 1951, the problem of Heterodera on tobacco was brought to the attention of A. L. Taylor of the Division of Nematology by B. F. Lownsbery of the Connecticut Agricultural Experiment Station. Since then, Taylor has reported (6), that the Connecticut nematode shows no morphological differences from Heterodera rostochiensis Wollenweber. Taylor has shown further that this nematode, which grows and reproduces on tobacco, also completes its life-cycle on tomato and that it does not reproduce on potato.

In addition to this work involving the Connecticut Heterodera, it was deemed advisable to reinvestigate the possible susceptibility of Nicotiana spp. to the Long Island, New York golden nematode, using a previously unexplored approach, i.e. to determine by means of stains whether or not golden nematode larvae enter tobacco roots and their eventual fate in the roots if entry is accomplished. A number of species and varieties of tobacco (listed below), made available to the authors through the cooperation of E. E. Clayton of the Division of Tobacco, Medicinal and Special Crops, U. S. Department of Agriculture, have been examined.

Methods

Tobacco seedlings were transferred to soil containing H. rostochiensis cysts, and to Heterodera cyst-free soil. In the latter case, infective H. rostochiensis larvae were introduced around the roots of each plant. All plants were maintained in pots in the Hicksville greenhouse. Roots were collected at intervals and fixed and stained in Fleming's Chromo-aceto-osmic Acid Formula (Weak). The roots were then washed and examined under the binocular dissecting microscope for the presence of stained H. rostochiensis larvae and developmental stages. The previously Heterodera-free soil from around the potted plants which had been exposed to larvae was washed through sieves and examined for the presence of mature saccate female nematodes.

Results

The roots of the following four commercial varieties of shade tobacco (which received special attention because the Connecticut Heterodera was found on shade tobacco) contained varying numbers of larvae at periods up to 47 days: Regular Shade, 200 larvae; Florida 301, 448 larvae; Comstock Spanish, 256 larvae; and Havana K-2, 352 larvae. Several of the larvae found in the roots were third stage forms within the second stage cuticles. Giant cells were not observed in the root tissue at nematode loci. Roots of these varieties, fixed 135 days after exposure, showed disorganized and decomposed larvae, apparently dead before fixation. Mature nematodes were not found.

All other examinations of soil balls and roots of the species and varieties listed below for the presence of mature female nematodes were negative during June 1952, after exposure periods ranging from 150 to 238 days.

Species and varieties of Nicotiana that were tested

Varieties of N. tabacum: Regular Shade, 402, Florida 301, Comstock Spanish, Havana K-2, Vesta 33, Golden Burley.

Species of Nicotiana: knightiana, paniculata, glauca, rustica var. pumila, rustica var. brasilia, otophora, setchellii, tomentosa, glutinosa, tabacum, tabacum-glauca tetraploid, palmeri, trigonophylla, alata, langsdorffii, longiflora, plumbaginifolia,

sylvestris, repanda, bigelovii, clevelandii, maritima, velutina, gossei, benthamiana, megalosiphon, occidentalis, debneyi, debneyi-tabacum tetraploid, debneyi-glauca tetraploid, angustifolia.

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DIVISION OF NEMATOLOGY INVESTIGATIONS, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, U. S. DEPARTMENT OF AGRICULTURE, HICKSVILLE, N. Y.

SUSCEPTIBILITY OF NICOTIANA SPP. TO THE GOLDEN NEMATODE, HETERODERA ROSTOCHIENSIS

W. F. Mai

The golden nematode, Heterodera rostochiensis Wollenweber, is a major pest of potatoes in all parts of the world where it is known to occur. Tomatoes are the only other commercially important crop plant on which this nematode has been shown to complete its life cycle. Triffitt (1929), Reinmuth (1929), Mai and Lownsbery (1948), Oostenbrink (1950), and Jones (1950) were unable to find developed females on the roots of Nicotiana tabacum or related species of Nicotiana when growing in heavily infested soil.

Taylor (1952) reported the finding by Dr. B. F. Lownsbery of a cyst-forming nematode attacking tobacco in Connecticut. This nematode was morphologically indistinguishable from the golden nematode of potatoes. Greenhouse tests showed tomatoes to be a host but no development occurred on potato roots. Evidence was obtained by Lownsbery (1952) that the tobacco cyst nematode strongly attacks the roots of several tobacco varieties, but develops less readily on tomato roots. He observed no development on potato roots.

Experiments

In 1946, seeds of 17 varieties of Nicotiana tabacum[1] were planted in heavily infested experimental plots located on Long Island. Two three-foot replications of each variety were planted in rows three feet apart. At intervals of one month, two months, and two and one-half months after planting, the roots of one third of the plants from each replication were removed from the soil and examined for the presence of swollen females on the roots. No females were observed on the roots of any of the tobacco varieties although numerous developed females were present on the roots of 39 varieties of tomatoes grown in the same field at the same time. No swollen females were evident on the roots of N. suaveolens or N. glauca growing in this field.

In 1950, studies were made on the following species: N. sylvestris, N. repanda, N. plumbaginifolia, N. rustica, N. bigelovii, N. langsdorffii, and N. nudicaulis. Ten plants of each species were grown in four-inch clay pots of heavily infested soil. The roots exposed when the intact soil balls were removed from the clay pots were carefully examined, and no swollen females were found.

Since the discovery of the tobacco cyst nematode in Connecticut, tobacco has been retested for susceptibility to attack by the golden nematode of potatoes. Three consecutive plantings of Regular Shade tobacco were grown in heavily infested soil over a period of 385 days with no evidence of developing females on the roots. Large numbers of hatched larvae were used as inoculum with negative results. In some of these tests larvae were added at different stages of development of the tobacco plants. During the summer of 1952, no swollen females could be found on roots of Regular Shade tobacco growing in heavily infested soil in the field.

Staining N. tabacum roots grown in heavily infested soil has shown that golden nematode larvae readily enter the roots. However, further development of larvae has not been observed. In one test in which tobacco roots were harvested at regular intervals, there were no more larvae inside roots after growing in infested soil for 80 days, than after 40 days.

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3. Mai, W. F. and Lownsbery, B. F. 1948. Studies on the host range of the golden nematode of potatoes, Heterodera rostochiensis Wollenweber. Amer. Pot. Jour. 25: 290-294.

[1]Big Orinoco, Jamaica Wrapper, Medley Pryor, Virginia Sun Cured, Deer Tongue, Improved White Stem, Bonanza, Lizard's Tail, Yellow Pryor, Sweet Orinoco, Virginia Bright, White Burley, Blue Pryor, Gold Dollar, Mammoth Gold, Improved Yellow Orinoco, and Hickory Pryor.

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INJURY TO SUGAR BEETS BY LIGHTNING[1]

R. D. Watson[2] and Ted Gessel[3]

In the arid irrigated areas where lightning injury to crops is less frequent than in the areas of high rainfall, the occurrence of a field spot that has all the appearance of a serious, rapidly-spreading disease is all the more alarming to field men and growers. This description of lightning injury with photographs is presented to aid in the identification of this type of injury on sugar beets. Fig. 1, 2, p. 488; Fig. 3, 4, p. 489.)

In the middle of June a field of beets was called to our attention in the Greenleaf area near Caldwell, Idaho. The grower described the spot as one in which the beets had been dying over a period of weeks, and the spot was becoming progressively larger. The area was circular, 40-feet in diameter (Fig. 1) and was located near a spot where the return soil from the previous crop had been dumped. The fact that this was near the returned soil led to the theory that some new trouble had been introduced into the field at that point.

The first evident symptom developed during the heat of an afternoon when apparently normal plants wilted (Fig. 2). Plants appearing to be normal were distributed in the spot where most of the plants were dead (Fig. 3), giving the appearance of a spreading disease.

When slightly affected roots were cut lengthwise the mildest evident symptoms were in the inner tissues. These central tissues were shrunken, leaving a pithy center, gray in color and quite dry (Fig. 4). More severely injured beets showed similarly killed tissue about three inches down from the crown, connecting the pithy center to the exterior of the sugar beets. This injury usually surfaced in a column of fine roots on the side of the beet.

In the injured beets this area became progressively blackened and disintegration caused by saprophytic or weakly parasitic organisms was in progress (Fig. 4). The wilting symptom developed in those sugar beets in which the blackening had advanced up the injured pithy interior of the beet to the crown. Thus the blackening, progressive wilting, and death of the sugar beets in the outer part of the circular area was due in part to secondary organisms that were able to take advantage of the unusual injury. The lower part of the tap roots of wilted beets remained sound (Fig. 4).

Beets in various stages of injury were collected and an attempt was made to isolate some pathogen from the beets. None of the isolates proved pathogenic on mechanically injured beets or seedlings. The beets collected from the field were ground fine and the ground tissue introduced into sterile soil in which sugar beet stecklings and seedlings were grown, but no disease developed.

The circular spot that developed in a field af Greenleaf was classed as due to lightning injury for the following reasons: this spot was similar to other reported cases of lightning injury; injury was most severe in the center of the circular spot and decreased outward to the edge; the outermost plants showed no symptoms at first but wilted and later died as a result of damage to the roots; the lower tap root usually remained sound where the plant wilted and died; finally, the nature of the internal injuries was typically that of lightning injury, i.e., a necrosis and collapse of tissues sometimes followed by secondary invaders. In addition, there was no evidence of pathogenic organisms either in the beet or the soil, since the replanted beets grew on the same rows quite normally through the remainder of the season and cultures or macerated tissue failed to yield any organism capable of causing damage to beet stecklings.

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AMALGAMATED SUGAR COMPANY, NAMPA

[1]Published with the approval of the Director of the Idaho Agricultural Experiment Station.
[2]Associate Plant Pathologist, University of Idaho.
[3]Research Department, The Amalgamated Sugar Company, Nampa, Idaho.

FIGURE 1. Lightning injury in sugar beet field. (See page 487.)

FIGURE 2. The edge of lightning-injured spot. (See page 487.)

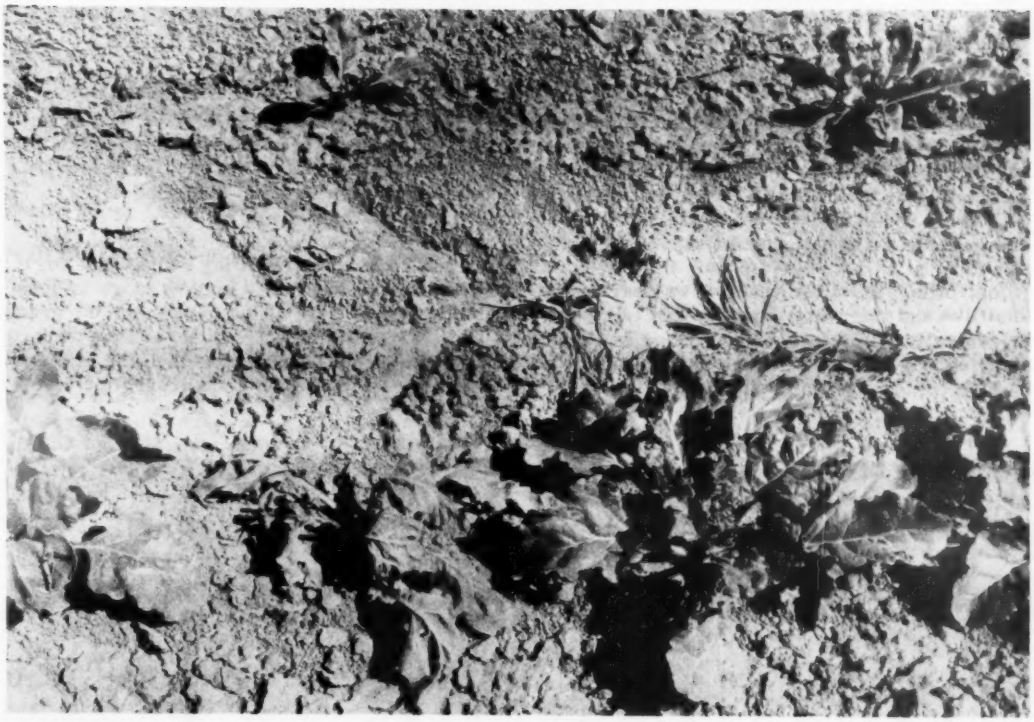


FIGURE 3. Close-up of row near edge of lightning-injured spot showing the wilted and apparently normal plants. (See page 487.)

FIGURE 4. Sectioned healthy and injured roots showing stages progressively toward the center of the lightning injured spot. (See page 487.)

WINDTHROW OF ROOT-ROTTED OAK SHADE TREES

W. A. Campbell and J. H. Miller

During a recent wind storm accompanying a thundershower at Athens, Georgia, two large oaks on the University of Georgia campus were windthrown. Both trees had had fruiting bodies of Polyporus dryadeus at the base for several years. The large roots of one tree were almost completely decayed (Fig. 1) as were most of the intermediate size roots of the other.

Polyporus dryadeus is responsible for widespread root rot in many of the trees of all the commonly planted oak species at Athens and elsewhere in Georgia. Positive evidence of root infection is indicated by the flat, thickened, yellow-brown to brownish fruiting bodies that develop, often year after year, at or near the base of affected trees. These may be more or less shelflike and may vary considerably in size when attached to the base of the trunk near the ground, or centrally stipitate when occurring on the ground and attached to a root. Observations over a period of years have shown that the amount of decay in the roots is related to the size of the fruiting bodies. Multiple fruiting at the base, and extending for some distance around the tree, is usually a sign of massive decay (Fig. 2).

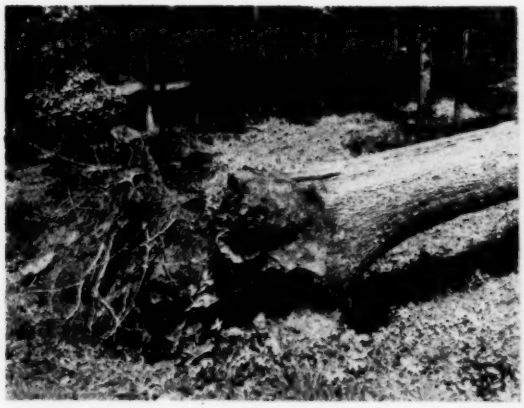


FIGURE 1. A white oak windthrown because its large roots were rotted by Polyporus dryadeus.

FIGURE 2. A large fruiting body of P. dryadeus at the base of a water oak.

Because of the weakened root system, windthrown hazard is great in yard and street oaks with roots extensively rotted by P. dryadeus. The presence of fruiting bodies of this fungus should be looked upon as indicators of a possible serious weakening of the root system.

DIVISION OF FOREST PATHOLOGY, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AND THE DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF GEORGIA, ATHENS, GEORGIA

UNUSUAL RECORDS AND OTHER BRIEF NOTES

TARGET SPOT ON SOYBEANS IN VIRGINIA

By Helen S. Sherwin

The target spot fungus, Corynespora cassiicola (Berk. & Curt.) Wei, previously reported as far north as Elizabeth City, North Carolina, was found on soybeans at the Virginia Truck Experiment Station, Norfolk, Virginia, October 8, 1952. Spotting was noticeably prevalent on leaves of the varieties Dortchsoy 31 and Roanoke.

DIVISION OF FORAGE CROPS AND DISEASES

SEVERE VIRUS DISEASE ON LADINO CLOVER IN VIRGINIA

By S. B. Fenne

During the past year letters were received from farmers from all parts of the State complaining that Ladino clover was not growing well and that the plants were stunted and failed to produce the abundant amount of grazing as in the past.

On October 22, Dr. K. W. Kreitlow, Plant Pathologist, U. S. Department of Agriculture; Dr. R. G. Henderson, Plant Pathologist, and Dr. G. M. Shear, Plant Pathologist of the Virginia Experiment Station; and S. B. Fenne, Extension Plant Pathologist, visited several farms in Hanover and Henrico Counties where complaints had been received concerning Ladino clover failure.

Earlier this season, the trouble with Ladino clover had been tentatively diagnosed as being caused by a virus. On this field trip it was definitely identified as a virus disease. The virus affecting Ladino clover is the same as the one found on alfalfa and on many other clovers commonly grown on these farms. After Ladino clover has been grown for several years, the virus apparently spreads from nearby leguminous crops that carry it but are apparently not damaged by it.

VIRGINIA POLYTECHNIC INSTITUTE EXTENSION SERVICE, BLACKSBURG, VIRGINIA

PLANT DISEASE NOTES FROM TEXAS

By A. L. Harrison

A meadow nematode found this past summer was causing considerable damage to localized spots in a corn field. Samples of corn roots and soil were submitted to A. L. Taylor, Division of Nematology, who tentatively identified the nematode as Pratylenchus zeae. A number of farmers complained of spots in their corn fields that appeared on the surface to be identical with the area sampled, but no detailed survey was made to determine the distribution or extent of the damage. As far as it is known, this is the first time that Pratylenchus zeae has been reported in Texas.

Downy mildew (Pseudoperonospora cubensis) has made its appearance this fall and has caused defoliation of volunteer watermelons and other small watermelon plantings, in spite of the fact that weather has been extremely dry since last spring except for one period early in September. Downy mildew was not observed during the spring season on watermelons or cantaloupes in the south central region of Texas.

We have also experienced an outbreak of the tomato russet mite (Phyllocoptes destructor) in our fall planting of tomatoes. This mite has been reported in Texas before but not in this area. At first we thought the plants were suffering from one of the streak diseases, but on close examination, under the binocular, the mites were found crawling on the leaves and stems of the affected areas.

TEXAS AGRICULTURAL EXPERIMENT STATION, YOAKUM, TEXAS

GETTING AT THE "MEAT" OF PLANT PATHOLOGICAL LITERATURE

By John A. Stevenson

To plan and carry out research in plant pathology and mycology demands a resonably complete and continuing background of what has been previously accomplished along similar lines by other workers. As in all fields of science, the literature on these subjects comes in an ever

increasing flood. Pertinent papers are not only exceedingly numerous, but widely scattered through a bewildering array of books, journals, proceedings, bulletins, and a general miscellany. They appear in many countries and in a confusing array of languages. Thorough indexing of the contents of this material is essential for phytopathological progress.

Efforts in this direction were initiated by the Federal Horticultural Board of the Department about 1916 when R. Kent Beattie commenced a host index of the world's phytopathological literature. This index was transferred to the Bureau of Plant Industry in 1927 and has been in progress in the Division of Mycology and Disease Survey since that time insofar as time and funds have permitted. In 1933 the indexing plan was broadened to include coverage of both hosts and causal organisms. In other words a fungus index was set up coordinate with the original host index.

These indexes present a unique series of detailed references to the literature on plant diseases and to the fungi in general. Though necessarily far from complete, they do contain an effective cross section of the published data in these fields. Reference cards as now made up provide the technical names of host and parasite, synonomy for fungus names, geographical distribution and an indication of the character of the technical material given in the article concerned in each case. They are used for general reference purposes and as an aid in plant disease and fungus identifications. Basic data from these files has been used in the preparation of a number of publications, including "Index of Plant Diseases in the United States," Foreign Plant Disease Manual," "Bibliography of the Fungi and Bacteria Associated with Tung," and "An Annotated List of the Fungi and Bacteria Associated with Sugarcane."

Work on these indexes, which had been for the most part in abeyance for some years, has been actively resumed and it is the hope not only that current literature will be covered as published but that progress can be made in working up the back log of earlier publications as yet unindexed which accumulated during the war years and the economy era which followed. The work is being carried on by Mr. Bernard Lipscomb, a skilled bibliographer and indexer, recently transferred to the Division from the Department Library. These indexes are always readily available for consultation by interested plant pathologists or others interested, and insofar as time permits inquiries by mail involving these files will be handled by staff members.

DIVISION OF MYCOLOGY AND DISEASE SURVEY

ANNOUNCEMENT: PLANT PROTECTION BULLETIN

The new Plant Protection Bulletin published monthly by the Food and Agriculture Organization of the United Nations contains reports on new or threatening plant disease and pest occurrences throughout the world, and notes on new control measures and other topics of interest. Persons who are interested in receiving it should write to Dr. Lee Ling, Plant Production Branch, Agriculture Division, Food and Agriculture Organization, Viale delle Terme di Caracalla, Rome, Italy.

The Plant Disease Survey is the official correspondent for the Bulletin in this country. Our task is to obtain information and transmit a summary report on plant diseases for publication each month. We would appreciate it if plant pathologists would send us suitable material from time to time for inclusion in our reports. Please mark it clearly so that we will know it is intended for the Bulletin. P. R. M.

THE INTERNATIONAL CODE OF BOTANICAL NOMENCLATURE

The long awaited revised edition of the rules of botanical nomenclature under the new title, International Code of Botanical Nomenclature, has been received. The code embodies the extensive changes voted by the Seventh International Botanical Congress held in Stockholm, Sweden in July 1950. The editorial committee set up by the Congress has effectively met the difficult problem of arranging in orderly and usable manner the involved and at times obscure directives of the Congress. The book has been published by the International Bureau for Plant Taxonomy and Nomenclature of the International Association for Plant Taxonomy and the Chronica Botanica Co. at Utrecht, Holland, with J. Lanjouw as chief editor.

A review of changes in the rules affecting the nomenclature of the fungi was presented in this Journal shortly after the Congress (Plant Disease Reporter 34: 327-333. 1950). However, since the several articles have been rearranged and renumbered in the new code, it will be worth while to review briefly features of particular concern to phytopathologists.

Article 8 presents the new term Taxon (plural Taxa) to apply to taxonomic groups of any rank. The term is used throughout the code and will prove useful in taxonomic publications. Section 2 (articles 18-22) establishes firmly the type method and Appendix I provides the recommended methods of determining types for Taxa of all ranks. The approved recommendation that in the cryptogams the types of pre-starting point authors may be accepted for species for which the first legitimate specific epithet is taken from Linnean or post-Linnean sources has apparently been ignored.

The starting point dates (Article 23) for lichens and myxomycetes (Linnaeus, Species Plantarum ed. 1, 1 May 1753) and for the Uredinales, Ustilaginales, and Gasteromycetes (Persoon, Syn. Meth. Fungorum, 31 Dec. 1801) have not been changed. For all other fungi Fries' Systema Mycologicum in all its parts including the Elenchus Fungorum (1828) constitutes the starting point, vol.1 of which is treated as having appeared on January 1, 1821. All fungi (except those based on Persoon) take their starting point with Fries if treated in any part of the work cited. For fungi not considered by Fries valid publication may occur in the work of any other author publishing after January 1, 1821.

Attention is called to Sections 5 and 6 covering conditions and dates of effective and valid publication. These two types of publication are defined in Article 10. Adequate descriptions and Latin diagnoses are required for new taxa. Illustrations and deposit of type material in recognized herbaria are recommended.

Article 69 is of direct concern to all dealing with fungus names and is quoted in its entirety:

> "In Ascomycetes and Basidiomycetes with two or more states in the life cycle (except those which are lichen-fungi), but not in Phycomycetes, the first valid name or epithet applied to the perfect state takes precedence. The perfect state is that which bears asci in the Ascomycetes, which consists of the spores giving rise to basidia in the Uredinales and of the chlamydospores in the Ustilaginales, or which bears basidia in the remaining Basidiomycetes. The type specimen of a state must bear that state. However, the provisions of this article shall not be construed as preventing the use of names of imperfect states in works referring to such states. The author who first describes a perfect state may adopt the specific epithet of the corresponding imperfect state, but his binomial for the perfect state is to be attributed to him alone, and is not to be regarded as a transfer.
>
> "When not already available, binomials for imperfect states may be proposed at the time of publication of a perfect state or later, and may contain either the specific epithet of the perfect state or any other epithet available."

It is much to be hoped that the new code will prove satisfactory to the majority of American phytopathologists and that its provisions will be followed in the interests of uniformity and stability in nomenclature. We can all agree with Dr. Lanjouw when he says "I wish to express the hope that we will not change our Code at every subsequent Congress. There are more important things to do! May the present Code obtain the approval of a very large number of botanists. The Editorial Committee is convinced of the shortcomings of this Code, but it hopes that it will be given a fair trial."

Nevertheless it may be noted that the American Phytopathological Society has appointed a temporary committee to deal with matters of fungus nomenclature which may need attention by the forthcoming Paris Congress. Proposals for corrections in the present code or for deletions or additions may be sent to any member of the committee. -- John A. Stevenson, Chairman, Nomenclature Committee, American Phytopathological Society.

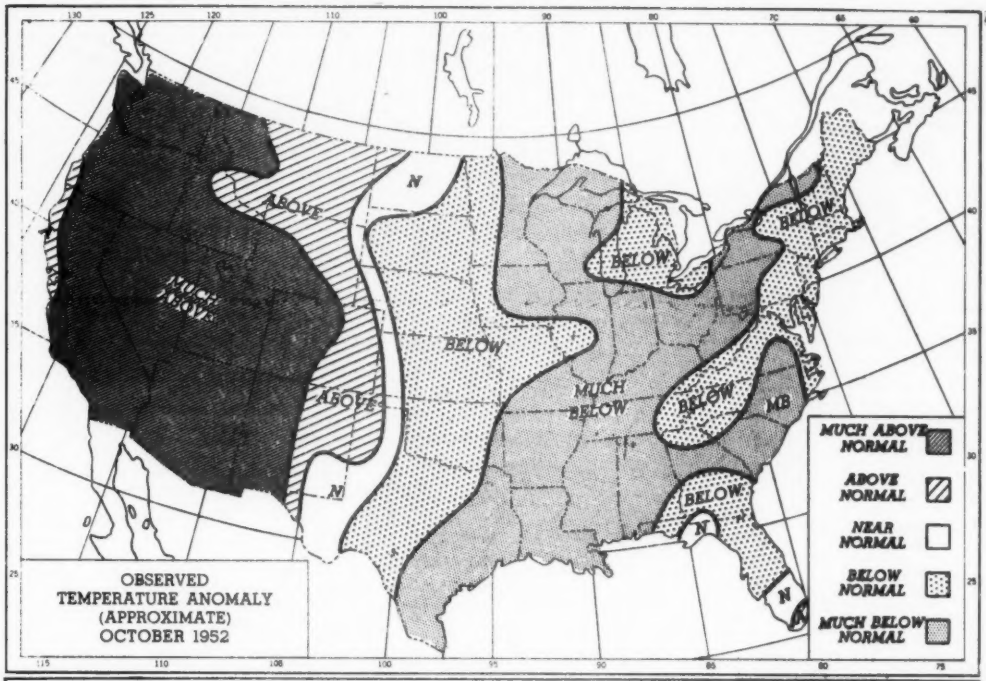
ATTENTION PLEASE!

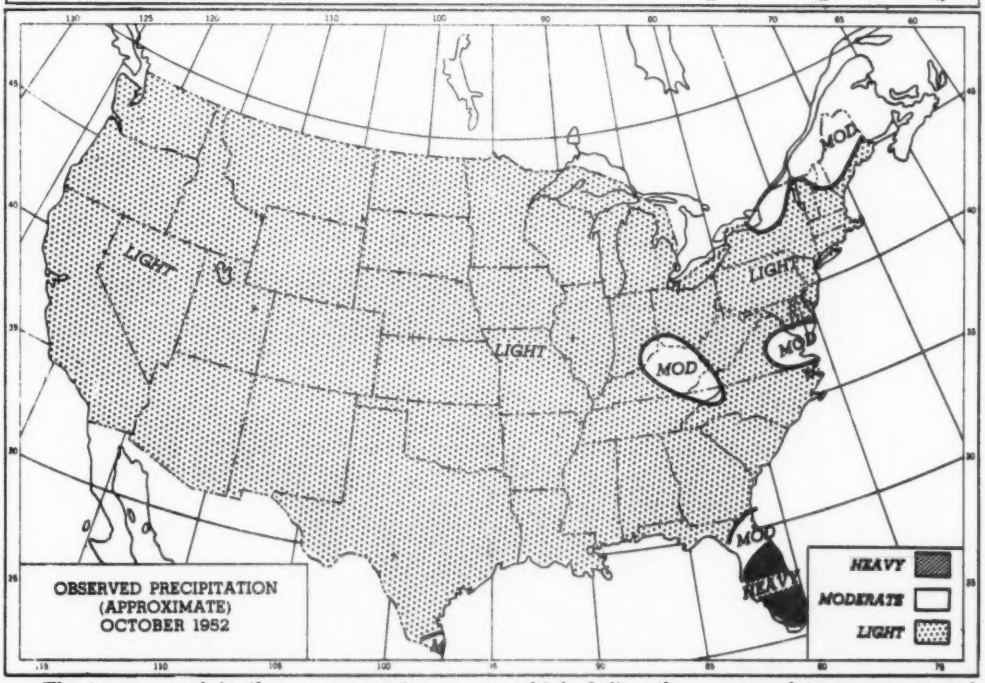
CONTRIBUTORS PLEASE NOTE

It is essential that the causes of diseases should be stated as specifially as possible in every article, even the briefest. Otherwise, uncertainty as to the identity of organisms or other factors involved may destroy part of the value of the article. Scientific names should be given for bacteria, fungi, and nematodes. A virus should be designated so that its identity is clear. Similarly with non-parasitic diseases, if a specific factor is known it should be mentioned. If the cause of a disease is not known, that should be stated too.

This might seem self-evident, and most of our contributors are as careful as could be desired. Occasionally, however, authors forget that not all readers will be so knowledgeable as themselves about the causes of the diseases they are discussing. Articles about control are apt to fail in this manner, even though the specific cause of the disease is important in control.

Please remember: Many of our readers are not plant pathologists. Not all pathologists are really familiar with diseases outside their own special fields. The Reporter has an international circulation, and as anyone knows who reads reports from other countries, it is hard to be sure what disease is meant unless it is clearly identified. -- P. R. M.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.

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